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(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit, Mich.

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Vol. LXXIII, N. S. Vol. 26 **May, 1928** **No. 1**

AT MINNEAPOLIS IN 1902

With the approach of the Minneapolis convention we frequently are asked questions about the meeting held in that city over a quarter of a century ago. Here are a few facts:

The thirty-ninth annual convention of the A. V. M. A. was held in Minneapolis, Minnesota, September 2-5, 1902. Dr. J. F. Winchester presided and Dr. S. Stewart was secretary. The five vice-presidents for 1901-02 were: Drs. Roscoe R. Bell, M. E. Knowles, W. H. Dalrymple, C. C. Lyford and Wm. Dougherty. Dr. Wm. Herbert Lowe was treasurer and is the only officer of the cabinet of eight who is still living.

The attendance was officially reported as 256. This number included 54 ladies. The number of members in attendance was 97. The balance included veterinarians who were not members, and a few who were designated as "other visitors." Headquarters were at the West Hotel and a surgical clinic was held at University Farm on the final day of the meeting. Dr. S. Stewart was elected president over Dr. A. H. Baker by a vote of 68 to 29.

Among the papers presented were the following: "Equine Periodic Ophthalmia," by Dr. M. Jacob; "Poisoning of Stock," by Dr. N. S. Mayo; "The Pathogenesis of Equine Pneumonic Emphysema," by Dr. A. H. Baker; and "Hemorrhagic Septicemia in Cattle," by Dr. S. D. Brimhall.

The membership of the A. V. M. A. in 1902, including the new members added at the Minneapolis meeting, was 453. Of this number 152 are on the membership roll at the present time.

EXECUTIVE BOARD ELECTIONS

Executive Board elections in two districts are now in full swing. The "primaries" came to an end on April 7 and ballots have been mailed to all paid-up members in the two districts. In District No. 2 there was a tie for fifth place, necessitating placing six names on the ballot. This is the third time that this contingency has arisen in recent years, and, as pointed out before, is not specifically covered by the By-laws.

Although the number of nominating ballots returned was rather small, the voting in the election proper is heavy, indicative of the interest being taken by our members since they have had the opportunity to look over the list of nominees. Four ex-presidents of the A. V. M. A. grace the tickets, three in District No. 3 and one in District No. 2. Practitioners are unusually well represented. Following are the nominees:

District No. 2

De Vine, J. F. — Goshen, N. Y.

Consultation and research work. Graduate of American Veterinary College, 1898. Joined A. V. M. A., 1899. Member of Committee on Abortion, 1919-25.

Hollingworth, W. G. — Utica, N. Y.

Veterinarian, Bureau of Health, Utica, N. Y. Graduate of American Veterinary College, 1884. Joined A. V. M. A., 1885. Member of Committee on Legislation, 1913-16; resident secretary for New York, 1918-19.

McNeil, J. H. — Trenton, N. J.

Chief, New Jersey Bureau of Animal Industry. Graduate of University of Pennsylvania, 1898. Joined A. V. M. A., 1899. Vice-president, 1907-08, 1909-10 and 1922-23.

Marshall, C. J. — Philadelphia, Pa.

Professor of Veterinary Medicine, University of Pennsylvania. Graduate of University of Pennsylvania, 1894. Joined A. V. M. A., 1895. Vice-president, 1903-04 and 1908-09; secretary, 1910-13; president, 1913-15.

Munce, T. E. — Harrisburg, Pa.

Director, Pennsylvania Bureau of Animal Industry. Graduate of University of Pennsylvania, 1904. Joined A. V. M. A., 1904. member of Executive Board, District No. 2, 1918-28.

Udall, D. H. — Ithaca, N. Y.

Professor of Veterinary Medicine and Hygiene, Cornell University. Graduate of New York State Veterinary College at Cornell University, 1901. Joined A. V. M. A., 1913. Member (1923-27) and chairman (1927-28) of Committee on Intelligence and Education.

DISTRICT NO. 3

AXBY, W. A. — Harrison, Ohio

Practitioner. Graduate of Ohio Veterinary College, 1895, and Cincinnati Veterinary College, 1905. Joined A. V. M. A., 1907. Member of Committee on Intelligence and Education, 1927-28.

BRUMLEY, O. V. — Columbus, Ohio.

Professor of Veterinary Surgery, Ohio State University. Graduate of Ohio State University, 1897. Joined A. V. M. A., 1919. Member (1920-21) and chairman (1926-27) of Committee on Necrology; member of Committee on History, 1923-25; member of Schmidt Memorial Committee, 1927-28.

MERILLAT, L. A. — Evanston, Ill.

Editor, North American Veterinarian. Graduate of Ontario Veterinary College, 1888. Joined A. V. M. A., 1893. Vice-president, 1906-07; secretary, 1916-17; president, 1924-25.

SIGLER, T. A. — Greencastle, Ind.

Practitioner. Graduate of Indiana Veterinary College, 1902. Joined A. V. M. A., 1909. Chairman of Finance Committee, 1913-15; resident secretary for Indiana, 1915-16 and 1922-23; representative to Horse Association of America, 1924-26; president, 1926-27.

WHITE, DAVID S. — Columbus, Ohio

Dean, College of Veterinary Medicine, Ohio State University. Graduate of Ohio State University, 1890. Joined A. V. M. A., 1905. President, 1920-21; member of Executive Board, District No. 3, 1923-28.

The polls will close June 7.

VETERINARIAN IN NEW FIELD

The Sioux Falls Serum Company, of Sioux Falls, South Dakota, has secured the services of Dr. Louis D. Mersch as advertising manager. It is believed that this is a brand new field for a veterinarian.

Dr. Mersch finished his veterinary course at Iowa State College, March 16. While in college he participated in many extra-curricular activities, which gave him considerable experience that will stand him in good stead in his new position. One of the honors won by Dr. Mersch as a student was his election to Sigma Delta Chi, professional journalistic fraternity. He was the first veterinary student to be so honored.

At the 1928 annual meeting of the Iowa Veterinary Medical Association, in Des Moines, Dr. Mersch took an active part in the publicity work connected with the meeting. Much of the splendid publicity secured in the daily and farm press, on that occasion, was the result of his efforts.

POULTRY DISEASE NUMBERS

Several letters have been received inquiring about the special poultry disease numbers of the JOURNAL that have been published. To make this information conveniently available for all our members, we give the four issues of the JOURNAL that have been devoted exclusively to papers on various poultry disease problems: May, 1925; February, 1926; February, 1927; February, 1928.

The four issues of the JOURNAL listed contained forty-eight papers on poultry diseases. In addition to the papers in these four special numbers, there have been twenty-three others published at various times, during the same period, making a total of sixty-one papers on avian pathology in about three years. These have covered practically every poultry disease of any importance at the present time.

ADD COPPER

Copper is now a candidate for recognition as one of the elements essential for proper nutrition, if we may judge from the results of investigations being conducted at the University of Wisconsin, by Dr. E. B. Hart and his associates. The experiments already conducted point very strongly toward copper as being an important factor in anemia. Iron has been pretty generally regarded as the element principally involved in anemia. It is known to be a part of the highly complex molecular formula of hemoglobin. However, the Wisconsin researches indicate that, although iron or its compounds are limiting factors, their effectiveness depends on the absence or presence of copper. The action of the latter may be in the role of a catalytic agent, assisting in the formation of hemoglobin without actually entering into its composition. It is hoped that further studies will clear up this question.

EASTERN STATES TUBERCULOSIS CONFERENCE

The Eastern States Tuberculosis Conference this year will be held at The Weirs, New Hampshire, June 19-20-21. Dr. E. A. Crossman, of Boston, is chairman of the Program Committee. A departure from the custom of former conferences will be made this year in that diseases other than tuberculosis will be discussed. The program will cover the sanitary production of milk; abortion, rabies, poultry diseases, etc. Dr. D. C. Lochead,

health officer of Rochester, Minn., will address the gathering on the subject of "Milk and Health." Dr. Crossman writes that extremely low hotel rates will be in effect and a large attendance is expected.

VETERINARIANS STUDY MILK CODE

The Standard Milk Control Code of the United States Public Health Service is being revised by Mr. Leslie C. Frank, sanitary engineer in charge, Office of Milk Investigations. At the invitation of Surgeon-General H. S. Cumming, the American Veterinary Medical Association has been asked to study the proposed revision and for that purpose President Hilty has seen fit to appoint a special committee as follows: Dr. George W. Grim, Ardmore, Pa., chairman; Dr. John P. Turner, Washington, D. C.; Dr. E. D. King, Jr., Valdosta, Ga.; Dr. J. B. Hollingsworth, Ottawa, Ont.; and Dr. Roy F. Leslie, Cleveland, Ohio.

VISITORS AT THE JOURNAL OFFICE

The JOURNAL office has been favored by visits from quite a number of out-of-town veterinarians during recent months. The list includes the following: Dr. Robert S. MacKellar, New York, N. Y.; Dr. N. S. Mayo, Chicago, Ill.; Dr. C. H. Stange, Ames, Iowa; Dr. John Reichel, Glenolden, Pa.; Dr. Reuben Hilty, Toledo, Ohio; Dr. B. J. Killham, Lansing, Mich.; Dr. Herman Konst, Ottawa, Canada; Dr. W. J. Embree, Chicago, Ill.; Dr. J. E. Shillinger, Washington, D. C.; Dr. W. L. Williams, Ithaca, N. Y.; and Dr. L. B. Sholl, East Lansing, Mich.

APPLICATIONS FOR MEMBERSHIP

Applications for membership have been coming in impressively. This month we are listing 26 for the first time and 57 are receiving the second listing. In the five issues of the JOURNAL published this year, January to May, inclusive, 175 applicants have been listed. The following table shows how 1928 compares with previous years, for similar periods:

	1925	1926	1927	1928
January	2	4	10	15
February	9	22	28	36
March	6	10	14	41
April	10	12	15	57
May	7	28	16	26
	34	76	83	175

Indications are that we will have 4000 members on the roll by the time the Minneapolis meeting convenes. All applications listed next month can be completed by August 1. Do not forget the method for handling applications now in effect:

Applications for membership shall be made upon blanks furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Applications must be accompanied by a membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Applications must be filed with the secretary and examined by him for correctness and completeness as far as available information will allow. After such approval by the secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

The amount that shall accompany an application filed this month is $8.33, which covers membership fee and dues to January 1, 1929, including subscription to the JOURNAL.

FIRST LISTING

BETZOLD, WILLIAM F. 819 M St., Sanger, Calif.
M. D. C., Chicago Veterinary College, 1905
Vouchers: W. L. Curtis and John L. Tyler.

BLAKE, WALTER G. P. O. Box 615, Greeley, Colo.
D. V. S. Colorado Agricultural College, 1911
Vouchers: Geo. H. Glover and I. E. Newsom.

BUTLER, HOMER CLAYTON Lock Box 456, Madelia, Minn.
D. V. M., Chicago Veterinary College, 1916
Vouchers: S. N. Kingrey and J. N. Gould.

CHENEY, DAVID W. 201 Patterson St., Ogdensburg, N. Y.
D. V. M., Cornell University, 1922
Vouchers: David F. Deming and C. E. Hayden.

CONOVER, ROBERT 624 Huron St., Toledo, Ohio
D. V. M., Chicago Veterinary College, 1920
Vouchers: Fred E. Anderson and Reuben Hilty.

FITZWATER, MAHLON CLAUDE Bloomingdale, Mich.
D. V. S., Grand Rapids, 1906
Vouchers: Edw. K. Sales and B. J. Killham.

FORD, IRA M. Edgerton, Minn.
D. V. M., Terre Haute, 1918
Vouchers: W. L. Boyd and R. Fenstermacher.

GAUNTT, I. G. Jasper, Ala.
D. V. M., Alabama Polytechnic Institute, 1915
Vouchers: C. A. Cary and C. C. Middleton.

GOLDEN, JOHN WESLEY Redwood Falls, Minn.
M. D. C., Chicago Veterinary College, 1895
Vouchers: E. A. Hewitt and R. Fenstermacher.

GORDON, REUBEN 400 W. Main St., Patchogue, L. I., N. Y.
D. V. M., Cornell University, 1925
Vouchers: P. A. Fish and C. E. Hayden.

GRADY, DANIEL THEODORE Windom, Minn.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: M. H. Reynolds and R. Fenstermacher.

GREAVES, HAROLD A. Glenwood, Minn.
D. V. M., Ohio State University, 1911
Vouchers: C. P. Fitch and R. Fenstermacher.

GREEN, MERLE CEDRIC Spring Grove, Minn.
D. V. M., McKillip Veterinary College, 1915
Vouchers: W. L. Boyd and R. Fenstermacher.

HANSON, R. E. Forest City, Iowa.
D. V. M., McKillip Veterinary College, 1910
Vouchers: C. H. Stange and John B. Bryant.

HATTERY MORTON 5714 Prairie Ave., Chicago, Ill.
M. D. V., McKillip Veterinary College, 1907
Vouchers: H. Busman and Otto Hornlein.

JOHNSON, CLARENCE HENRY 1061 S. Smith Ave., St. Paul, Minn.
D. V. M., McKillip Veterinary College, 1916
Vouchers: G. E. Totten and Irvin Owens.

LAMOREAUX, MORRIS FLORANCE Comstock Park, Mich.
D. V. M., Michigan State College, 1927
Vouchers: E. T. Hallman and B. J. Killham.

LARSON, GEORGE A., JR. Breckenridge, Minn.
D. V. M., Chicago Veterinary College, 1920
Vouchers: C. P. Fitch and R. Fenstermacher.

MEYERS, SIDNEY RALPH Mountain Lake, Minn.
D. V. M., Grand Rapids, 1911
Vouchers: C. P. Fitch and R. Fenstermacher.

NOONAN, ALBERT J. 5733 S. Peoria St., Chicago, Ill.
M. D. V., McKillip Veterinary College, 1908
Vouchers: H. Busman and Otto Hornlein.

OSTENDORF, ALFORD T. Red Lake Falls, Minn.
D. V. M., McKillip Veterinary College, 1913
Vouchers: C. P. Fitch and R. Fenstermacher.

RUNYAN, BRUCE 124 E. McCreight Ave., Springfield, Ohio
D. V. M., Cincinnati Veterinary College, 1915
Vouchers: H. W. Feldwish and D. M. Swinehart.

THOMPSON, JOHN EINO 245 W. Harvey St., Ely, Minn.
D. V. M., Ohio State University, 1919
Vouchers: E. A. Hewitt and R. Fenstermacher.

VANCURA, THOS. W. New Prague, Minn.
D. V. M., McKillip Veterinary College, 1916
Vouchers: W. L. Boyd and R. Fenstermacher.

VOLLMAR, OTTO GEORGE Montgomery, Minn.
D. V. M., McKillip Veterinary College, 1912
Vouchers: R. Fenstermacher and C. P. Fitch.

WOLF, ARVINE J. Bolivar, Ohio
D. V. M., Ohio State University, 1907
Vouchers: Reuben Hilty and S. W. Brown.

Applications Pending

SECOND LISTING

Beckmann, Wilhelm E., 3604 Loughborough Ave., St. Louis, Mo.
Bowers, Wm. O., Spring Valley, Ohio.
Brobeck, Ivan J., Richwood, Ohio.

Brown, James Thomas, 510 E. D. St., Belleville, Ill.
Bunnell, Wm. A., Miamitown, Ohio.
Clark, Wm. R., Wauseon, Ohio.
Dabbelt, A. H., 504 E. Main St., Coldwater, Ohio.
Deem, Arthur W., R. F. D. No. 3, Delaware, Ohio.
Dilts, Charles R., Napoleon, Ohio.
Ewing, Harry E., 77 W. Northwood, Columbus, Ohio.
Faust, J. Henry, 7628a S. Broadway, St. Louis, Mo.
Fitzpatrick, J. C., Amelia, Ohio.
Fogle, Allan E., 2478 Neil Ave., Columbus, Ohio.
Gilbertson, Henry, Madison, S. Dak.
Gill, Joseph H., 721 Second St., Portsmouth, Ohio.
Hopkins, Troy Syrus, Box 2012, Ancon, C. Z.
Jacobi, Van S., Greenville, Ill.
Keef, Earl M., 301 Wholesale Terminal Bldg., Los Angeles, Calif.
Kickbusch, Frank, 342 Superior St., Milwaukee, Wis.
Lange, J. V., Mechanicsburg, Ohio.
Leasure, Elden Emanuel, Kansas State Agricultural College, Manhattan, Kans.
Ludwig, Wm. F., Barnesville, Ohio.
Lukens, William R., Hillsboro, Ohio.
Lynch, Leland C., 1737 Tytus Ave., Middletown, Ohio.
McConkey, Charles W., Napoleon, Ohio.
Meier, Frederick D., 838 27th Ave., Milwaukee, Wis.
Meyer, Leo P., Convoy, Ohio.
Meyer, Oliver A., East Alton, Ill.
Michael, Leo B., Collinsville, Ill.
Miller, George O., Kenton, Ohio.
Morris, Fred J., Morral, Ohio.
O'Brien, W. S., Ryan, Iowa.
O'Harra, William G., Camp Chase, Ohio.
Owen, Roger N., 416 S. Prospect St., Ravenna, Ohio.
Quirin, Charles P., Smithton, Ill.
Parmer, H. W., Ida Grove, Iowa.
Priest, Frederick, 88 S. Third St., Newark, Ohio.
Ragsdale, Howard LeRoy, Oxford, Pa.
Redden, Elwin M., Box 259, Norfolk, Nebr.
Redman, W. B., Boxholm, Iowa.
Rolighed, Marvin H., Appleton, Minn.
Satorius, George R., Athens, Ill.
Simpson, Harvey Lewis, 739 4th St., San Diego, Calif.
Smiley, Ray S., Cromers, Ohio.
Spindler, John L., Ashville, Ohio.
Stanley, Carl Black, Belleville, Ohio.
Stevenson, Lionel, Ontario Veterinary College, Guelph, Ont.
Stortz, Lewis J., Edwardsville, Ill.
Swale, Floyd S., 2882 Humboldt, S., Minneapolis, Minn.
Todd, R. C., 107 Washington St., Delta, Ohio.
Turpen, Stephen A., 357 W. George St., St. Paul, Minn.
Underwood, Forrest A., Vaughnsville, Ohio.
Westerberg, Ralph Victor, Simsbury, Conn.
Williams, Newcomb L., College Corner, Ohio.
Wilson, Harold E., Arenzville, Ill.
Woods, Leonard B., Carrollton, Ill.

COMING VETERINARY MEETINGS

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. & 103rd St., New York, N. Y. May 2, 1928. Dr. C. P. Zepp, Secretary, 128 W. 53rd St., New York, N. Y.

Connecticut Veterinary Medical Association. New Britain, Conn. May 2, 1928. Dr. C. H. Patchen, Secretary, Milford, Conn.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. May 2, 1928. Dr. W. G. Oliver, Secretary, 3821 Arizona St., San Diego, Calif.

Chicago Veterinary Society. Great Northern Hotel, Chicago, Ill. May 8, 1928. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.

Hudson Valley Veterinary Medical Society. Catskill, N. Y. May 9, 1928. Dr. J. G. Wills, Secretary, 122 State St., Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Detroit Mich. May 9, 1928. Dr. H. Preston Hoskins, Secretary, 716 Book Bldg., Detroit, Mich.

Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo. May 15, 1928. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Anglees, Calif. May 16, 1928. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Central Michigan Veterinary Medical Society. Otsego Hotel, Jackson, Mich. May 23, 1928. Dr. W. N. Armstrong, Secretary, Concord, Mich.

California State Veterinary Medical Association. San Diego, Calif. June 11-12, 1928. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Texas, State Veterinary Medical Association of. State College, Texas. June 18-22, 1928. Dr. D. Pearce, Secretary, Leonard, Texas.

Eastern States Tuberculosis Eradication Conference. The Weirs, N. H. June 19-20, 1928. Dr. E. A. Crossman, Chairman, Program Committee, 2001 Customhouse Bldg., Boston, Mass.

Michigan State Veterinary Medical Association, Michigan State College, East Lansing, Mich. June 26-27, 1928. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing.

Missouri Veterinary Medical Association. Carthage, Mo. June 26-28, 1928. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

North Carolina State Veterinary Medical Association. Greensboro, N. C. June 27-28, 1928. Dr. W. T. Scarborough, Secretary, 320 S. Blount St., Raleigh, N. C.

THE PHYSIOLOGY OF MILK FEVER. II*

By PIERRE A. FISH, *Ithaca, N. Y.*
Cornell University

HYPOGLYCEMIA AND NORMAL BLOOD SUGAR STANDARDS

In a former paper[1] attention was called to data which seemed to controvert the theory of a reduced amount of sugar in the blood (hypoglycemia) as the causative factor of milk fever, as advocated by Widmark and Carlens,[2] Auger[3] and others. Additional data by Hayden[4] and by Moussu[5] strengthen the evidence against a hypoglycemia.

The necessity for a normal working standard for the sugar in the blood is self-evident; some confusion has already resulted from the use of different standards. The range in human and canine blood has been given as 80 to 120 milligrams of glucose per 100 cc of blood. That of bovines is less. Widmark and Carlens and Auger give 80 mg. per 100 as the standard for dry cows and 60 mg. per 100 for milk cows. Moussu with a group of eleven animals (3 oxen, 3 dry cows and 5 cows in milk) obtained an average of 61 mg. per 100. Hayden and Sholl,[6] using the Folin-Wu method, obtained an average of 51.75 mg. per 100. Their average was determined as a result of 75 examinations from 44 cows. They were "good specimens in so far as metabolism and general physical condition are concerned." Most of the animals were in milk, but a few in the dry condition were included in the average.

A standard obtained by averaging the results from a group of animals, taken at a given time, has an undoubted value; but it is believed that a more reliable standard can be obtained by continuing the examinations for a long period of time (a year) and obtaining the average amount for the period. We are following this plan with a monthly blood-test on a group of five cows. Four were pregnant when the experiment began; three were in milk and two were dry. After an approximate six-month period (October to June), three produced calves and the fourth calf was born two months later. The fifth cow was not bred until December and is now in the pregnant condition. After ten months of the experiment, the average of 48 individual tests gives 45.52

*Presented at the sixty-fourth annual meeting of the American Veterinary Medical Association, Philadelphia, Pa., September 13-16, 1927.

mg. of sugar per 100 cc of blood. The maximum obtained in any one animal was 70 mg., taken on the day the calf was born, and the minimum 30 mg.

This average and that obtained by Hayden are noticeably lower than those given by the European investigators. In general, it would appear that the amount of blood sugar in the bovines is about one-half that of man or dog. We believe that a range of 40 mg. to 60 mg. per 100 is a reliable standard for the average normal cow. Although sugar is one of the most important constituents of the blood, it is not apparently confined to narrow limits as to its amount. Auger, in agreement with Widmark and Carlens, believes that reduction of the blood sugar below 40 mg. or 30 mg. causes hypoglycemic disturbances. We have found, in apparently normal cows, the blood sugar as low as 30 mg. without evidence of this disturbance.

Amount of Blood Sugar in Dry Cows and Milk Cows

Widmark and Carlens, examining heifers and dry cows, obtained an average of 80 mg. per 100; in milk cows the average was 60 mg. per 100. The lower average in the latter was supposed to be due to the using up of the glucose of the blood by the udder in manufacturing lactose. Auger agrees with the above figures. In our experiment the two dry cows gave an average, during the dry period, of 44.89 mg. per 100 (av. 14 tests) and two cows in milk, an average of 46.68 mg. per 100 (av. 20 tests). Moussu's data show an average of 56 mg. per 100 for three dry cows, an average of 53.2 mg. per 100 for three males (oxen); and for three cows (pregnant) in milk 71.5 mg. per 100 and two cows (non-pregnant) in milk 64.2 mg. per 100. In our experiment four of the cows were pregnant when the experiment began and the fifth became so three months later.

It is realized that data from a larger number of animals are desirable, but it is nevertheless significant that independent groups of workers, contrary to the findings of Widmark and Carlens, find in the jugular blood a slightly greater amount of sugar in the milk cows than in the dry cows.

Influence of Pregnancy upon the Amount of Blood Sugar

Although there may be conditions associated with a normal course of pregnancy which causes the amount of blood sugar to fluctuate, it is believed that they cause transitory rather than continuous changes. Monthly examinations of the sugar for six months in two cows, prior to calving, showed an average of

47.5 mg. per 100; examinations for three months after calving gave an average of 46.7 mg. per 100. Diet and, perhaps, seasonal conditions are probably more important factors in regulating the sugar supply of the blood. A study of the sugar curve of the five cows shows that, during the ten-month period, the amount of sugar fluctuated very little during October, November and December. There was a uniform decrease in sugar during January and February, an increase in March, a decrease in April, a rise for May and June and a decline in July. The curves are suggestive of seasonal changes, as the animals were kept under the same conditions. They were in pasture from about May 20 to November 1 and received a small amount of grain and hay in addition. From November to May they were stabled and allowed access to a paddock for exercise, and received a larger proportion of grain and hay than during the summer months. In all of our tests the blood was taken from the jugular vein and tested by the Benedict reagent with the Folin-Wu filtrate.

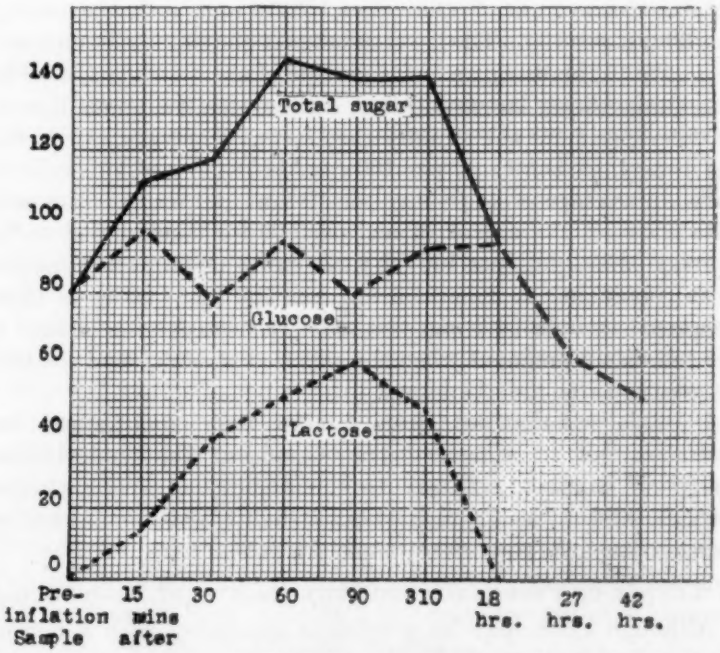

FIG. 1. (Case 9.) The curve for the total sugar represents the results as ordinarily obtained by the Folin-Wu test. After the fermentation method has been applied, the glucose disappears and the lactose remains. The Folin-Wu method is again applied to determine its amount. The glucose curve is determined by subtracting the lactose from the total sugar. Note that the pre-inflation sample of blood contains no lactose; but after inflation the lactose is quite markedly increased and has disappeared within 18 hours.

Amount of Blood Sugar Before and After Udder Inflation

We have been able to confirm, on milk-fever cases, the observations of Widmark and Carlens on *healthy* cows, that inflation of the udder increases the amount of sugar in the blood. We have also been able to confirm their hypothesis that this increase is due to the absorption of lactose from the mammary gland. In the former papers of Fish[1] and of Hayden,[4] the method of Folin and Svedberg for differentiating between glucose and lactose in the blood by the process of fermentation has been described.

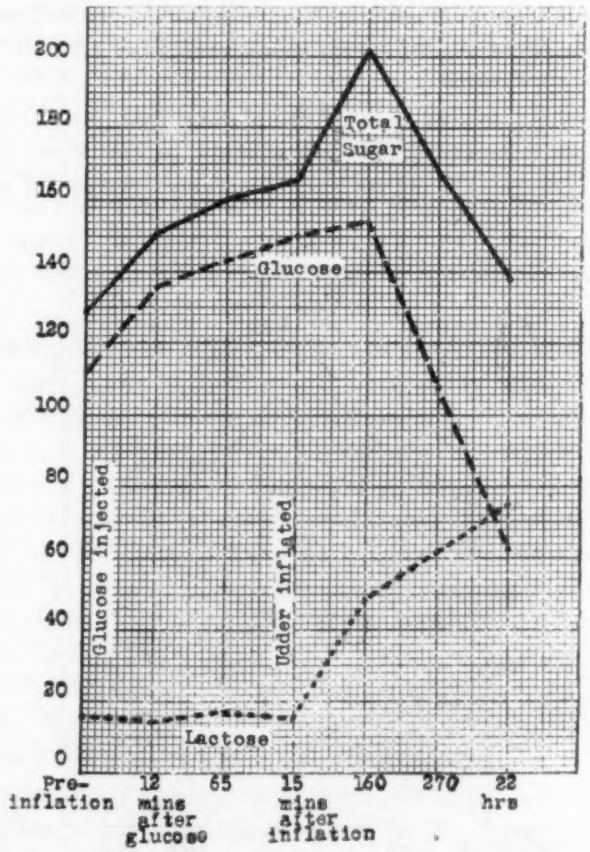

Fig. 2. (Case 10.) This case of milk fever was the only one in our series which showed the presence of lactose in the blood before the udder was inflated (lactosemia). Immediately after the pre-inflation sample of blood was taken, 20 grams of glucose were injected into the jugular vein. The udder was not inflated until 90 minutes later. Note that during this period the amount of lactose remained practically unchanged, while the amount of glucose increased, probably as a result of the injection. After the udder was inflated there was a marked rise of the lactose in the blood and but little change in the glucose until the third hour, after which it decreased and was approaching normal at the 22nd hour.

Two of the cases were referred to in Fish's paper and five additional ones were reported by Hayden. Out of a total of seventeen cases to date, the method has been applied to seven and we believe the results are reliable. In only one (case 10) of the seven cases, was any lactose found in the blood before the udder was inflated and this small amount was considerably increased after inflation. In this case the udder was considerably distended with milk before treatment was given and it may be assumed that lactose absorption had been going on before the pre-inflation test was made. In the six other cases there was no evidence of lactose in the blood until after the udder had been inflated. It usually appeared within fifteen minutes and the amount varied with individual cows.

Our tests have also enabled us to show that the increase of sugar after inflation is not due entirely to the absorption of lactose (lactosemia). There is also a minor increase of glucose

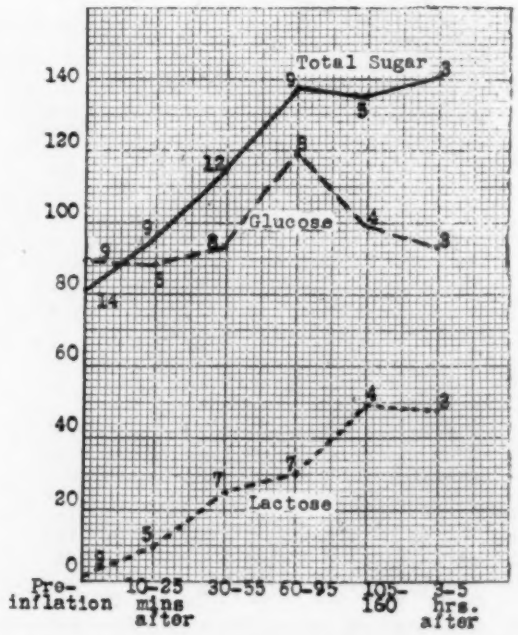

FIG. 3. (Averages.) This chart represents the averages of the sugar determinations. There were no pre-inflation samples from three cases. The lactose determinations were not made on the first five cases. In some cases only one or two lactose tests were made. The figures along the curves represent the number of cases from which the average was made. The curves indicate that the glucose reaches its maximum in 1 to 1½ hours and the lactose in 2 to 4 hours.

(glycosemia). In cases 6 and 9 our data were complete enough to show that in the former, as compared with the pre-inflation sugar (glucose), the average increase of the total sugar for 60 minutes was due to an increase of 84.5 per cent of lactose and 16.1 per cent of glucose. In the latter (case 9), over a period of 310 minutes, there was an average increase of 83.3 per cent of lactose and 15.9 per cent of glucose. Whether or not this percentage is constant, we have not as yet sufficient data to show. This minor increase in the percentage of glucose after inflation is perhaps accounted for by the fact that the gland is temporarily defunctionized and the glucose that had hitherto been used in the production of lactose has now an opportunity to accumulate in the blood. In some cases it was not infrequently found that the post-inflation glucose occasionally dropped to a lower level than the pre-inflation glucose—there was more or less fluctuation. Udder inflation of dry animals has no effect upon the sugar content of the blood, according to Auger. He inflated the udder of a three-year-old dry goat, making sugar tests before and after inflation and found that neither hyperglycemia nor glycosuria appeared.

There seems to be considerable variation in the return of the glucose and lactose to their normal levels in the blood after inflation. In case 6, after one and three-fourths hours, the glucose had fallen to 42.7 mg. (normal limit) but the lactose was still high (32 mg.), although receding. In case 8, after 17 hours, no lactose was found but the glucose was high (103 mg.). In case 9 the lactose had disappeared within 17 hours but the glucose was still high (93.2 mg.). At the 46th hour it was normal (51.3 mg.). In case 10, after 23 hours the glucose was approaching normal (62 mg.) but the lactose was the highest of our readings (75 mg.). This cow had received an intravenous injection of 20 grams of glucose. In case 13, after four hours the glucose and lactose were both high, 79.2 mg. and 35 mg., respectively.

The amount of sugar present in the blood, when treatment is begun, has a very direct bearing on the hypoglycemic theory. In this connection also it is obvious that a normal standard for comparison is of importance. Widmark's standard of 60 mg. per 100, Moussu's of 61 mg. per 100, Hayden and Sholl's of 51.75 mg. per 100, and our recent average of 45.52 mg. per 100 for a ten-month period show fairly wide variations.

In the fourteen cases of milk fever reported by Hayden and Sholl, they found that the average amount of sugar present in the "first samples" was 99.42 mg. per 100. In Hayden's later report[4] of fourteen additional cases, the "first samples" showed an average of 94.45 mg. per 100. Unpublished data by Little and Keith,[9] on five cases of milk fever in which the blood samples were taken before udder inflation, showed an average of 93.2 mg. per 100. Moussu reports the average for five cases "before treatment" as 62.6 mg. per 100. The average of fourteen of our recent cases "before treatment" was 81.5 mg. per 100. In connection with our average it is interesting to note that the sugar readings were relatively low in all the cases occurring between October 6, 1926, and January 24, 1927, the average of 8, "before treatment," being 64.5 mg. per 100, agreeing quite closely with Moussu's average. The minimum was 45 mg. and the maximum 80 mg. From January 27 to May 26, the readings were higher, giving an average of 104.1 mg.; the minimum being 80 mg. and the maximum 127.8 mg. It is hoped in the course of time that enough data may be obtained to determine if such a marked difference may be due to seasonal conditions.

The "first samples" referred to in Hayden's papers were taken just after the udder had been inflated and perhaps explains why his figures are higher than those obtained by Moussu and by us in our later series "before treatment." It is difficult to construe the above data with a condition of hypoglycemia in milk fever.

Auger states that Widmark and Carlens, experimenting on eight subjects (cows and goats), found a sugar increase, after inflation of the udder, with a maximum at 40 to 60 minutes and a rather rapid return to normal at about the ninetieth minute. Presumably these figures refer to healthy animals. In a number of our milk-fever cases the sugar did not return to its normal level until after a number of hours and in one case not until the second day.

To illustrate the increased amount of blood sugar following udder inflation, we have taken the average of twelve cases when the sugar was at its maximum (30 to 60 minutes after inflation) and find it to be 133.14 mg. per 100, an increase of 63.1 per cent over the *pre-inflation* (before treatment) sugar. About 84 per cent of this increase appears to be due to the absorption of lactose into the blood. Reckoning from a normal standard of blood sugar at 51.75 mg. per 100, the total increase would be 157.27 per cent. The greatest amount of total sugar found in the blood

of individual cases occurred in cases 10 and 14. Both of these animals had received injections of glucose. Case 10 received 200 cc of a 10 per cent glucose solution (20 grams of glucose). Four hours after the injection the Folin-Wu test gave 202.2 mg. per 100. Case 14 received an injection of 6 ounces of glucose in one quart of water (180 grams to 1 liter) and about one hour later the test showed 312.2 mg. of sugar per 100 cc of blood. These figures are probably far above the "renal threshold" and a marked glycosuria might be expected.

The Glucose Treatment

The injection of glucose solutions for curative purposes was evidently devised in connection with the hypoglycemic theory, for the purpose of restoring the normal amount of sugar to the blood and tissues. In a true hypoglycemia such a treatment is most logical and proper. If the blood sugar, however, is normal or higher than normal, as the figures of Moussu and ourselves show, such treatment would not, apparently, be indicated. Moussu applied the glucose treatment to three cows without apparent benefit. In the first cow he injected 30 grams of glucose subcutaneously. As there was no change in the symptoms in two hours, the udder was inflated and the patient recovered in four hours. In cow 2, the same amount of glucose was injected subcutaneously. There was no change in six hours. The udder was then inflated and the cow was standing in three hours. In cow 3, twenty grams of glucose were injected intravenously. There was no change during 17 hours. A second injection of forty grams was given subcutaneously. There was no improvement and the animal was sacrified.

In our case 10, 200 cc of a 10 per cent solution (20 grams) of glucose were injected in the jugular vein. As there were no observable changes after one and one-half hours, the udder was inflated and improvement followed.

Some reports have been made from various parts of this country in connection with the use of a commercial preparation in which the dosage is 6 ounces of glucose to a quart of water (180 grams per liter), intravenously administered. The reports are variable, some claiming favorable effects and others obtaining no benefit. In our own clinic this treatment has been used in two or three cases *after* the udder had been inflated. Apparently there has been no detriment and possibly some slight advantage.

In Widmark and Carlens' original paper,[2] reference is made to the curative effect of glucose injections. They report its use in four cases. A sterilized 5 per cent solution of glucose was injected intravenously (jugular or mammary veins) to the extent of three to five liters. This would mean the use of from 150 grams to 250 grams of actual glucose. One cow was in coma; in another, no coma was present; another was nearly unconscious and the fourth was unable to rise but at times was able to raise her head. In each case after the injection the cow was on her feet in an hour or less. After another hour the udder was inflated as they were unable to guarantee that the favorable effect of the injection would be lasting.

In comparing this method with the others previously mentioned the outstanding difference is the larger bulk of the solution used, and in some instances a greater amount of glucose. It is estimated that there are about 15 grams of glucose in the total blood of the average cow and on this basis a dose of 20 grams would seem reasonable. On the other hand an excessive amount of glucose in the blood would not necessarily be serious, as the excess, not used by the tissues, would pass over into the urine. The difference in results may be associated with the difference in the bulk of the solutions used as well as the glucose content. Approximately, the average cow may be considered to contain about 30 liters of blood in its body. The injection of 3 to 5 liters of fluid into the blood means an increase of 10 to 17 per cent of its volume and this may reasonably be expected to have a general stimulating effect, not only on the circulation itself but on some of the eliminating channels as well.

Intravenous Injection of Normal Saline

In connection with the effect of increasing the volume of the blood, we carried out an experiment on a normal Guernsey cow, December 28, 1926, using 4 liters (1 gallon) of normal (0.9 per cent) physiological saline solution (NaCl.). The cow was giving only three quarts of milk at the time and was milked once a day. The period of injection covered two hours. A small amount of the solution was lost in the process but we are sure that 3½ liters, or a little more, got into the circulation. The cow urinated and defecated a number of times during the experiment and it was noted that the feces were more fluid than normal. Apparently both kidneys and bowel were stimulated to greater activity.

A sample of blood was taken before the injection and gave a sugar reading of 49.1 mg. per 100. Rather to our surprise, it was found that four later samples, taken during the period of injection, showed an increased amount of sugar as follows: 1 hour, 71.7 mg.; 1¼ hrs., 67.9 mg.; 1½ hrs., 87 mg.; 1⅔ hrs., 83.6 mg.; and 4¾ hrs. from the beginning of the experiment a return to normal, 45.1 mg. The 1½-hr. amount of sugar (87 mg.) represents an increase of 77 per cent above the initial reading, an increase equal to that found in a number of our cases of milk fever. The injection represented the introduction of about 36 grams of sodium chlorid into the blood, but we have no knowledge that this salt has any glycogenic function and doubt that it is directly concerned in the production of sugar. Being an isotonic solution, the saline solution offers no insult to the blood, whereas pure water in this amount, if injected rapidly, might produce hemolysis. Slow injection, especially of a large quantity of fluid, is desirable, as there is a possible danger of dilation of the right ventricle if too rapidly introduced.

In a case of milk fever, the stimulation of the kidneys and bowel, as indicated in our experiment, ought to be a favorable factor; furthermore, the exchange of fluid to and from the blood and tissues, washing out the latter and probably promoting the process of oxidation, are additional favorable considerations and may assist in explaining the curative effects reported by Widmark and Carlens. A five per cent solution of sugar is likewise an isotonic solution; but, aside from this quality, it is not impossible that the glucose introduced may serve a purpose in connection with the oxidation processes following the stimulating action of the increased volume of blood.

Three samples of urine were also collected from this cow, each of which gave reduction tests indicating the presence of sugar. Unfortunately no urine sample was obtained before the experiment was begun.

The Effect of Udder Inflation upon Blood Pressure

Seitter,[8] experimenting upon normal animals (goats and cows), induced a lowered blood-pressure by the use of chloral hydrate and amyl nitrite. A Riva-Rocci apparatus was used in determining the blood-pressure. In the goats the normal blood-pressure, at the femoral artery, ranged from 196.7 to 211 mm. Hg. In the cows he found a pressure in the coccygeal artery ranging from 193.7 to 203.8 mm. Hg. A goat with a normal

pressure of 206.2 mm. and a temperature of 39°C. was given 5 grams of chloral hydrate per os and in half an hour showed complete narcosis. The pressure had fallen to 156.5 mm. and the temperature to 38.5°C. The udder was now inflated and a half-hour after the inflation the pressure had risen to 181.3 mm. and the temperature to 38.9°C. Three and one-half hours after udder inflation, both pressure and temperature had returned to normal. The same goat was used again five days later; this time without udder inflation—as a control experiment—the same amount of chloral hydrate being given. The initial pressure was 211 mm. Hg. and the temperature 38.9°C. After narcosis the pressure fell to 158 mm. and the temperature to 38°C. The blood-pressure and temperature gradually rose as the animal recovered, and reached normal six hours after the administration of the chloral. Udder inflation caused a return to normal pressure and temperature two hours earlier than by natural recovery.

Similar experiments were tried upon another goat and a cow with similar results. In the cow, inflation brought a return to normal in three hours; in the control experiment the normal pressure and temperature was not reached until seven hours. Data are wanting relative to such experiments upon actual cases of milk fever, but clinical evidence points to a condition of low blood-pressure. Three factors in the experiments show an analogy to the conditions found in numerous cases of milk fever: unconsciousness (coma), low blood-pressure and subnormal temperature. All of these are apparently improved by the simple inflation of the udder and it seems reasonable to assume that inflation will produce results in milk-fever cases similar to those in the experiment animals.

Urinary Ammonia

In Bulletin 160, Kentucky Agricultural Experiment Station, February, 1912, Healy and Kastle[10] point out analogies common to milk fever and eclampsia. Parturition is believed to be the most important factor. Quoting Hess (Schw. A., 1905) it is stated that "in 170 cases he did not observe it (milk fever) during, nor preceding labor, nor did he observe it later than 96 hours following labor." Exceptionally, however, cases have been reported, by others, before calving and also later than the 96-hour period.

These authors devoted especial attention to the urine. As a result of 100 examinations of the urine of the dairy cow, covering a period of three winter months and three summer months, they have established the following as a normal standard.

Color: Colcrless to deep yellow, clear, either no precipitate or a slight flocculent, or granular and occasionally a heavy white precipitate of calcium sulphate or triple phosphates.

Reaction: Alkaline, neutral or amphoteric.

Specific gravity: 1.014.

Hippuric acid: 1.17%.

Ammonia: 0.0009%.

Urea: 1.06%.

Total nitrogen: 0.58%.

Albumen and sugar: Absent.

Microscopical: Crystals of calcium sulphate, calcium oxalate, triple phosphates, ammonium urate, amorphous urates, squamous epithelial cells and irregular vegetable cells.

The urine from three cases of milk fever showed albumin present in all. The total nitrogen was increased; hippuric acid, decreased; urea, increased; ammonia, markedly increased. Hyaline, granular and a few epithelial casts and leucocytes were present.

The authors believe that the disease is due to a toxin elaborated in the udder, as the result of its own metabolism preceding normal milk-production, and that the success of the modern treatment is due to preventing, by means of pressure, the absorption of this toxin seems most highly probable.

If a mammary toxin is the causative agent, it may be assumed that milking out the udder would bring relief; but this procedure, according to others, favors the onset of milk fever.

Their evidence is based, in part, on the injection of the colostrum from milk-fever cases into the abdominal cavity of guinea pigs. Ten cc of the colostrum was used and death usually resulted in five or six days. The postmortems revealed acute parenchymatous nephritis with interstitial hemorrhages, acute parenchymatous hepatitis with interstitial hemorrhages, acute degeneration of the cells of the adrenal cortex, with complete destruction of the medullary cells, and interstitial hemorrhages. Five cc of colostrum from healthy cows or of fresh milk similarly injected into guinea pigs, although causing some reaction, did not cause fatal results.

In analyzing their data it is found that in two of their cases the ammonia content of the urine was forty times above normal; the other case showed only a trace. In one of our cases (13) an ammonia determination revealed its presence to the extent of 0.028 per cent, which is fairly close to their determinations: 0.037 and 0.035 per cent and much higher than the normal content of 0.0009 per cent.

Horvath[11] found that injections of ammonia (or urease) into rabbits caused a rise in the content of the glucose of the blood. This is suggestive in connection with the presence of ammonia in cases of milk fever and may have a bearing on the increased amount of the glucose found in the blood of such cases.

The Nitrogenous Constituents of the Blood

Hayden[15] seems to have been the first to have noted, in addition to a hyperglycemia, a changed nitrogenous condition of the blood

Table I—*Averages of blood analyses (mg. per 100 cc of blood), after Hayden*

	Sugar	Total Non-Protein Nitrogen	Urea	Uric Acid	Creatinine	Chlorids
Normal (Average of 75 tests)	51.75	30.11	11.18	2.06	1.37	516.1
1st samples milk fever (Average of 27 tests)	95.82	37.34	17.02	2.25	1.31	472.7
2nd samples, after treatment (Average of 9 tests)	68.44	38.24	14.04	2.19	1.11	515.62

in milk-fever cases as compared with the normal. The data on blood findings in table I, slightly revised, are taken from his work.

Hayden states:

> An average of the first samples of blood taken in milk fever indicates a rather marked hyperglycemia. The second set of samples which are taken after treatment (at intervals sufficient that the animals had shown recovery from the attack) still shows hyperglycemia but there is quite a marked decrease in the sugar content of the blood after udder inflation.
>
> There is an increase in total non-protein nitrogen in both sets of samples. The average for the second set is higher than that of the first. There is an increase in the urea in both sets but in this case the first shows the greater increase. Urea is a part of the total non-protein nitrogen and it makes up a larger portion of the non-protein nitrogen in milk fever than it does in the normal animal.

It is of interest to note that the non-protein nitrogen and urea still remain high after the apparent recovery of the patient. Although both of these constituents show higher readings than the normal, the proportion of urea in the non-protein nitrogen

has returned to normal, *i. e.*, in the normal, urea comprised 37 per cent of the non-protein nitrogen and in the "second samples" 36.7 per cent. In the "first samples" it is 45.5 per cent.

The urea of the "first samples" is 52.2 per cent higher than the urea of the normal animals and in the "second samples" is still 25.5 per cent higher. The total non-protein nitrogen of the "first samples" shows an increase of 24 per cent, and of the "second samples" 27 per cent increase over the normal. The creatinine shows a decrease of 4.3 per cent in the "first" and of 19 per cent in the "second" samples. The chlorids show some decrease in the first samples and the uric acid 9.2 per cent in the "first" and 6.3 per cent increase in the "second" samples. It would appear than even after the apparent recovery of the animal, there is, nevertheless, a delayed recovery of the nitrogenous metabolism.

The Endocrine System

It is now quite well established that, in one way or another, the pancreas (insulin), adrenals, thyroid and pituitary glands contribute directly or indirectly to the equilibrium of the sugar supply in the body. They may, therefore, be more or less involved in the altered glucose content in the blood of milk-fever cases.

Recently attention has been directed toward another group of endocrine glands—the parathyroids, which may have a more direct relation as a causative factor in milk fever. It is generally accepted that the parathyroids are concerned with, or regulate, the calcium metabolism of the body. Another function attributed to them, but not so frequently mentioned, is the regulation of the amount of the nitrogenous substance known as guanidin.

Halliburton[12] states: "When we remember that the creatine of muscle is methylguanidin acetic acid we see where guanidin originates, and in bringing about the destruction of this substance the parathyroids exercise a controlling influence on muscular metabolism and maintain muscular tone." According to Paton and Orr,[13] guanidin ($CNH(NH_2)_2$ is a near ally of urea $CO(NH_2)_2$.

Dryerre and Greig[14] suggest certain symptoms observed in milk fever analogous to those seen in animals deprived of their parathyroids, or those in which a parathyroid deficiency exists, particularly with reference to the guanidin function.

Among the typical signs of guanidin poisoning they refer to: preliminary excitement, dilation of the pupil, increased irrita-

bility of the neuro-muscular mechanism giving rise to muscular tremors and spasms, interference with gait and finally collapse. There is a lessened sensibility of the sensory nerves, abolition of the swallowing and coughing reflexes, an accelerated pulse due to a lowered tone of the cardio-inhibitory fibers, cessation of defecation and micturition to the depressing action upon the sympathetic system, while the toxic effects of guanidin upon the secretory activities of the kidneys result in the suppression of urine. The arrestment of the involution of the uterus and the lowered blood-pressure could be accounted for by the concentration of the guanidin being sufficient to paralyze all plain muscle.

The relationship of the adrenal glands to the sympathetic system is well known; and evidence indicates that the circulation of adrenalin, in sufficient amount, in the blood stream plays an important role in the rate of oxidation in the tissues. If the supply of adrenalin is subnormal, there will be a lessened destruction of the toxins which, in connection with a parathyroid deficiency, increases the toxemia.

They believe that inflation of the udder causes pressure sufficient to develop stimuli strong enough to stimulate reflexly the outflow of adrenalin and this may hasten the normal processes of oxidation.

Disturbance of the calcium metabolism is not lost sight of. This may act concurrently with adrenalin deficiency. Calcium is a normal constituent of colostrum and milk and through this channel the normal amount in the body may be reduced. Inflation, by checking the secretion of milk, will conserve the supply of calcium in the blood. With an increased amount of adrenalin, oxidation is hastened, the toxins are burnt up and blood-pressure is raised. Their hypothesis includes numerous points of physiological interest, and experimental work bearing on some of these points with their results is promised later.

Guanidin Experiment

On April 5, 1927, we had an opportunity to try the effects of this substance on a Holstein heifer weighing 405 kilograms (980 lbs.). She had aborted, February 4, producing a calf about eight months along. This was her second calf and she was now giving five quarts of milk in the morning and two quarts in the afternoon. Her temperature at 10 a. m., prior to the experiment, was 101.4° F. At 10:25 she was injected intramuscularly (gluteal muscles, left leg) with 200 cc of a solution containing 60.75 grams

of guanidin carbonate, a dosage of 0.15 grams per kilogram. Twenty-five minutes were consumed in introducing the solution, which was alkaline in reaction. A sample of blood was taken immediately after the injection. She was placed in a box-stall and within a half-hour showed marked signs of restlessness, lying down and getting up seven times during the following hour. The respirations were increased. At 12 o'clock the temperature had fallen to 101°F. At 1 p. m. it was 100.8° and there were evident muscular tremors (tetany) which were confined to the left hind quarter. At 3 p. m. the temperature had fallen to 100.5°; 3:20 p. m., second sample of blood. At 5:40 p. m. the cow was down and did not get up when approached. The temperature had risen to 101.8°F. An occasional slow twitch of the muscles in the parotid region (both sides) was observed.

TABLE II—*Results of blood tests (mg. per 100 cc of blood) following injection of guanidin carbonate*

	JUST AFTER INJECTION	4½ HRS. AFTER INJECTION	28½ HRS AFTER INJECTION	46 HRS. AFTER INJECTION
Sugar	44.8	56.1	52.6	50.6
Urea	11.3	10.4	14.5	12.4
Total non-protein nitrogen	18.0	19.5	22.8	21.0
Total non-protein nitrogen, less urea	6.7	9.1	8.3	8.6
Calcium	12.0	13.0	11.0	12.0
Refractive index of serum	1.3495	1.3500	1.3500	1.3491

April 6 (next day), 9 a. m., temperature 102.3°F. The heifer was standing up and looking bright. She gave her usual two quarts of milk the night before but the following morning gave only half her usual amount. At 3:25 p. m., 5 cc (100 units) of parathormone (Lilly) was injected intramuscularly. April 7, 9 a. m., temperature 101.8°F. Apparently normal.

In all, four samples of blood were taken for examination: (1) immediately after the injection of the guanidin carbonate; (2) 4½ hours later; (3) 28½ hours; and (4) 46 hours. The last sample was taken 17½ hours after the parathormone (McCollip's extract of the parathyroid glands) had been injected. The results of the blood tests are given in table II.

While the observed signs may apply to other conditions than milk fever, some are nevertheless suggestive: the restlessness at the onset, subnormal temperature, respiratory disturbance and muscular tremors. The blood-tests showed a moderate increase in the sugar, urea and total non-protein nitrogen, and agrees to some extent with the conclusions of Hayden[15] whose evidence was based on averages obtained from the study of 27 cases of milk fever.

A comparison of the percentages based on Hayden's results with those of the guanidin experiment is given in table III. The figures of the first blood-test are taken as the normal of the animal and the percentages of the guanidin experiment are based on them. The data cover the average of the three days of the experiment; two days of which the animal appeared to be entirely normal. Hayden's data cover a shorter period.

TABLE III—*Comparison of the percentages according to Hayden with those of guanidin experiment*

	First Samples (27) (%)	Second Samples (9) (%)	Guanidin Experiment (%)
Sugar increase	85.1	32.2	18.7
Urea increase	52.2	25.5	9.7
Total non-protein nitrogen increase	24.0	27.0	17.2
Non-protein nitrogen (less urea) increase	7.3	27.8	29.2

The difference seems to be principally one of degree. In each the same constituents were increased in amount and the same prolonged disturbance in the nitrogenous metabolism appeared. The most marked effect of the guanidin appears to be upon the non-protein nitrogen other than urea.

The calcium constituent of the blood showed little appreciable change, even after the administration of the parathormone. Our results are suggestive of a relatively mild effect from the guanidin. If larger or repeated doses could be given, or if a cow showing a deficiency of blood calcium could be used, there would probably be more complete and satisfactory effects.

Guanidin experiments were also conducted on frogs and guinea pigs. In the frogs there resulted twitching of the toes, and in the guinea pigs "shivering" suggestive of tetany. The injections of the guanidin carbonate were made intra-abdominally.

Hypocalcemia in Milk Fever

The relation of the parathyroid glands to the calcium metabolism of the body has already been referred to. Little and Wright, testing the blood of six healthy cows, obtained as an average 10.01 mg. of calcium per 100 cc of blood. Meigs gives the normal values as varying from 9 to 11 mg. per 100 cc of plasma. In eight cows "down" with milk fever, Little and Wright found the average to be 5.14 mg., with a range of 4.1 to 7.1 mg. A second test, made after the cow returned to normal, gave an average (6 cases) of 9.61 mg., ranging from 8.1 mg. to 10.45 mg. Four additional cases are reported separately in which the average of the four when the cows were "down" was 5.41 mg. and when normal (24 hours to 3 weeks) the average was 7.6 mg. These cases occurred upon the same farm and are noteworthy in connection with the low *normal* value of the blood calcium concentration. The animals were also slow in their recovery from the disease and it is suggested that dietetic factors may play an important part in the causation of milk fever.

A calcium deficiency and, perhaps, quite as important, a deficiency of vitamin D, in the diet may very reasonably be expected to have a predisposing influence as factors.

We have thus far applied the calcium test to only three cases of milk fever, in which the blood samples were taken before the udder was inflated, and found the average of the three to be 4.8 mg. of calcium, thus confirming the work of Little and Wright so far as cases "down" with the disease are concerned. The limits of time required for the blood calcium to return to the normal amount, during or after recovery, are not very definitely defined in Little and Wright's paper and our own cases are too few to throw much light on the subject. In our own case 14, the pre-inflation calcium was 6 mg.; after 1 hour and 25 minutes it was 5.6 mg. In case 15, the pre-inflation calcium was 3 mg.; in two hours it had increased to 7.5 mg. In case 16, no blood sample was taken at the onset of the disease; the udder was inflated and ten and one-half hours after the first inflation, the cow not doing well, a second inflation was given. Immediately after this inflation, a blood sample was taken and showed 11.3 mg. of calcium. Fifteen and one-half hours later, another blood sample showed 9.6 mg. of calcium. Both amounts represent normal limits and presumably were reached as a result of the first and subsequent inflations. Case 17 showed a pre-inflation calcium of 5.5 mg.; one hour later, 5.75 mg.

The return of the blood calcium to its normal amount is apparently a relatively slow process. There is probably no contribution to the calcium of the blood from the mammary gland similar to the hyperglycemia (lactosemia) following inflation, where there is evidence that lactose passes quickly into the circulation. The gradual return of the calcium to normal is apparently due to the prevention of its further loss in the milk by the inflation of the udder, with, we may assume, an improvement in the tone of the parathyroid glands and their power to regulate calcium metabolism.

A hypocalcemia theory seems to afford a more satisfactory explanation for certain of the symptoms of milk fever than does that of a hypoglycemia. There is a point of analogy, however, in the two theories, in that with the onset of lactation a new channel is provided for the loss of calcium as well as glucose from the body. Up to the time of the birth of the calf, there has been a drain on the calcium resources of the mother to develop the offspring (fetal skeleton, etc.) and with the new and increased demand for calcium in the colostrum and milk, especially in heavy milkers, the calcium reserve of the body may be dangerously depleted and the breakdown occurs. It is conceivable that even before lactation begins, the calcium reserve may become overtaxed in the development of the calf, especially if there is a deficiency of calcium in the diet; and thus account for the exceptional cases of milk fever occurring before the calf is born, or in the exceptionally late cases of the disease, the continued drain of the calcium in a heavy milk-yield, coupled with an inadequate supply of calcium in the food, may bring about disaster.

Little and Wright,[9] as an example of the drain of calcium from the body in the production of colostrum, estimate that a cow weighing 1200 pounds contains 100 pounds of blood and the percentage of calcium in the blood is 0.01, or the weight of calcium in the total volume of blood is 0.01 pounds (approximately 4.5 grams). They estimate the weight of one gallon of colostrum at 10 pounds and the calcium in the colostrum at 0.2 per cent, and the weight of the calcium in a gallon at 0.02 pounds (approximately 9 grams). The secretion of a half-gallon of colostrum would be sufficient to deplete the blood of all its calcium, supposing there was no reserve store in the body from which the blood might replenish its content. They also point out that in their milk-fever cases, the fall in the calcium content of the blood always ran parallel with the severity of the symptoms.

Cameron[17] states that cow's milk averages 120 mg. of calcium per 100 cc; a quantity 10 to 12 times higher than that found in the blood, and indicates the power possessed by the mammary gland in concentrating that amount from the 9 to 11 mg. present in the blood. Quoting Meigs, it is stated that a cow during 133 days of lactation lost 1300 grams (approximately 2.7 lbs.) of calcium, estimated at 19 per cent of its total body calcium, the loss being continuous; the milk output was about twenty pounds per day. There is, correspondingly, marked storage during the dry period. McCollum and Simmonds[18] state that of all the elements in the body, calcium is present in the blood in most constant amounts.

The endocrine influence in the control of important bodily functions is now too well established to be ignored: insulin regulates sugar metabolism; thyroxin, the iodin metabolism; and the parathyroids, the calcium metabolism, although vitamin D and the ultra-violet rays also have claims for consideration in this matter, as well as a possible guanidin function.

Cases of Milk Fever

Concise reports of thirteen cases have already been recorded in the papers of Fish[1] and of Hayden.[4] Four additional cases are herewith presented:

Case 14: March 23, 1927. Medium-size pure-bred Jersey cow, 11 years old; normal parturition; heavy milker. Temp., 98.4°; pulse, 75; resp., normal, except for expiratory groan. Animal shivered considerably (tetany), and was covered with a blanket. Three samples of blood were taken. Six ounces of glucose dissolved in one quart of water were injected into the jugular vein just before the second blood sample was taken. Recovery.

Case 15: March 26, 1927. Six-year-old Holstein cow. Found sick in the morning. Lying on right side, with head on left flank. Had been covered with a heavy blanket. Temp., 93.6°; pulse, feeble (80?). A blood sample was taken and the udder inflated; 10 cc of camphorated oil injected intramuscularly. Seen 1¾ hours later, she appeared brighter. Temp., 94.4°. Another blood sample taken and another blanket advised. Recovery.

Case 16: April 6, 1927. The cow was staggering about, apparently coming down with the disease, at 9:30 a. m. Temp., 98°. The udder was inflated but no blood sample was taken at this

time. The cow did not improve. In the evening she was in a semicomatose condition. Temp., 101°; pulse, 48; resp., slow. Udder inflated again at 8 p. m., 10½ hours after the first inflation. A blood sample was taken immediately after the second inflation. The next day (Apr. 7), at 11:30 a. m., 15½ hours after the first sample, another specimen of blood was obtained, and after this, glucose was administered. Recovery.

TABLE IVa—*Results of the blood examinations (mg. of sugar and calcium per 100 cc of blood) of case 14, March 23, 1927*

	PRE-INFLATION	25 MINUTES AFTER INFLATION (JUST AFTER INJECTION OF GLUCOSE)	85 MINUTES AFTER INFLATION (1HOUR AFTER INJECTION OF GLUCOSE)
Total sugar	101.6	122.0	312.2
Glucose	101.6	89.5	262.0
Lactose	0.0	32.5	50.2
Calcium	6.0	6.0	6.0
Refractive index of serum	1.3472	1.3480	1.3480

TABLE IVb—*Results of the blood examinations (mg. of sugar and calcium per 100 cc of blood) of case 15, March 26, 1927*

	PRE-INFLATION	110 MINUTES AFTER INFLATION
Total sugar	113.4	174.9
Glucose	113.4	119.1
Lactose	0.0	55.8
Calcium	3.0	7.5
Refractive index of serum	1.3470	1.3480

Case 17: May 16, 1927. Grade Holstein, 10 years old. Calved during the night. Was "down" in the morning. Had partially cleaned. Head on flank. Temp., 98.9°. Appeared dull. Udder inflated. Seen one hour later, the temperature was 100.5° and the udder again inflated. Two blood samples were taken and after this, 25 grams of glucose in 50 cc of solution were given intravenously. Recovery.

Tables IVa, IVb, IVc and IVd give the results of the blood examinations.

In two cases there was a rise in the refractive index of the serum after inflation. In the third case there was no pre-inflation sample but a sample taken 10½ hours after the first inflation gave figures comparable to the two other post-inflation cases. In the fourth case there was no change in the pre- and post-inflation samples.

Discussion

In our opinion, none of the seventeen cases examined by us has shown a true hypoglycemia. The lowest amount of blood

Table IVc—*Results of the blood examinations (mg. of sugar and calcium per 100 cc of blood) of case 16, April 6, 1927*

	Pre-inflation	10½ Hours After Inflation	26 Hours After Inflation
Total sugar	No blood samples taken	198.4	71.1
Glucose		174.8	63.2
Lactose		23.6	7.9
Calcium		10.5	9.6
Refractive index of serum		1.3481	1.3489

Table IVd—*Results of the blood examinations (mg. of sugar and calcium per 100 cc of blood) of case 17, May 16, 1927*

	Pre-inflation	1 Hour After Inflation
Total sugar	87.3	85.2
Glucose	87.3	82.1
Lactose	0.0	3.1
Calcium	5.5	5.75
Refractive index of serum	1.3488	1.3488

sugar in our series was 45 mg. (case 4) and is within our normal range. The majority of the cases showed an increase in the amount of the sugar.

The increased amount of sugar found in the blood after inflation, we believe we have successfully demonstrated, by the aid of the Folin-Svedberg fermentation test, to be due, for the most part, to the absorption of lactose from the mammary gland, and the minor increase of the glucose after inflation is due to the fact

that it is no longer used by the gland for the production of lactose and thus has an opportunity to accumulate in the blood.

As far as our work has gone (three cases) we have been able to confirm the observations of Little and Wright, that a deficiency of calcium is present in the blood of cows "down" with milk fever. Their work does not give definite information as to the length of time required for the calcium to rise to the normal level; nor have we sufficient data to throw light on that point.

TABLE V—*Results of sugar examinations (total sugar mg. per 100 cc of blood) 17 cases*

CASE	DATE	PRE-INFLATION	LENGTH OF TIME AFTER INFLATION								
			10–25 MINS.	30–55 MINS.	60–95 MINS.	105–160 MINS.	3–5 HRS.	10–12 HRS.	17–18 HRS.	21–27 HRS.	42 HRS.
	1926										
1	10–7	68.7	109.6	137.5							
2	10–10	70.7									
3	10–14	62.2	81.3	131.1							
4	10–23	45.0	73.3	87.1							
5	10–29	77.8	111.5	108.0	99.6	87.0					
6	12–31	62.7	77.7	113.4	119.4	74.7					
	1927										
7	1–13	49.0	60.0	64.0	62.1	R					
8	1–24								114.13*		
9	1–24	80.0	111.6	116.8	114.6	140.0	140.0		93.2	63.5	51.3
10	1–27	127.8	151.0†	160.0	165.3‡	202.2	168.0			137.6	
11	2–3	114.2		132.0	145.6						
12	2–18			101.3§	133.6						
13	2–18	80.6	90.0	113.2			114.2				
14	3–23	101.6		122.0‖	312.2						
15	3–26	113.4				174.9					
16	4–27							198.4		71.1	
17	5–16	87.3			85.2						
Average		81.5	96.2	115.5	137.5	135.76	140.7	198.4	103.66	90.7	51.3

R = Re-inflated.
*Average of three tests in one hour.
†Injection of 20 grams of glucose immediately after pre-inflation sample of blood was taken.
‡Udder inflated 90 minutes after glucose injection.
§This sample of blood was partially coagulated.
‖Six ounces of glucose is one quart of water were injected.

The indications are that it rises, after defunctionizing the gland by inflation, and while it is not being used by the gland, it accumulates gradually to the normal amount.

Using the Clark-Collip modification of the Kramer-Tisdall method, we have obtained as an average of 35 tests from nine *normal* cows, 11.7 mg. of calcium per 100 cc of blood. This is somewhat higher than that of Little and Wright (10.01 mg.), who

made their determinations from the ash of the blood. Ours were made upon the serum, and the presence of protein material and possibly some inexpertness in the earlier tests may explain our higher average.

Although Dryerre and Greig, so far as we know, were the first to offer plausible suggestions connecting the symptoms of milk fever with a parathyroid deficiency, especially in relation to guanidin intoxication, their suggestions were purely theoretical. The work of Little and Wright seems to be the first to offer positive evidence that the parathyroids are incriminated so far as calcium metabolism is concerned. Whether a hypocalcemia

TABLE VI—*Results of glucose determinations (mg. per 100 cc of blood)*

CASE	DATE	PRE-INFLATION	LENGTH OF TIME AFTER INFLATION								
			10–25 MINS.	30–55 MINS.	60–95 MINS.	105–160 MINS.	3–5 HRS.	10–12 HRS.	17–18 HRS.	21–27 HRS.	42 HRS.
	1926										
6	12–31	62.7	59.5	74.9	72.6	42.7					
	1927										
7	1–13	49.0	60.0	64.0	53.01						
8	1–24	—	—	—	—	—	—	—	114.13*		
9	1–24	80.0	97.77	77.5	94.24	79.3	92.5	—	93.2	63.5	51.3
10	1–27	112.5†	135.80	142.85	150.08‡	153.88	106.83	—	—	62.6	
11	2–3	114.2	—	111.78	110.4						
12	2–18	—	—	101.3	133.6						
13	2–18	80.6	90.0	84.2	—	—	79.2				
14	3–24	101.6	—	89.5§	262.0						
15	3–26	113.4	—	—	—	119.1					
16	4–6	—	—	—	—	—	—	174.8‖	—	63.2	
17	5–16	87.3	—	—	82.1						
Average		89.03	88.61	93.25	119.75	98.74	92.84	174.8	103.66	63.1	51.3

*Average of three tests in one hour.
†Twenty grams of glucose injected.
‡Udder inflated.
§Six ounces of glucose injected.
‖Just after second inflation.
—No material available at period indicated.

can explain all of the symptoms of milk fever is problematical and remains to be determined. Our single guanidin experiment on the cow is too incomplete to afford much evidence, but the blood picture points to some extent toward that of milk fever. The blood calcium, however, was not affected by the guanidin.

That tetany (muscular tremors) may be induced by a hypocalcemia is now pretty well established. That tetany can be induced by guanidin has been demonstrated by Watanabe[19] in rabbits, and in our own experiments. Whether the latter is just

a pharmaceutical effect, or whether some other explanation is necessary, has not been determined. The two forms of tetany do not seem to be identical. In guanidin tetany Watanabe finds that unlike the tetany induced by thyro-parathyroidectomy, it is not abolished by the administration of calcium.

Healy and Kastle[10] appear to have been the first to note a marked increase in the urinary ammonia in cases of milk fever. Our test, on a single case, was confirmatory of their observations. Watanabe in his guanidin experiments noted a great increase in ammonia in rabbits, and this observation would seem to lend support to the view that guanidin may be a factor in milk fever.

TABLE VII—*Results of lactose determinations (mg. per 100 cc of blood)*

CASE	DATE	PRE-INFLATION	LENGTH OF TIME AFTER INFLATION								
			10–25 MINS.	30–55 MINS.	60–95 MINS.	105–160 MINS.	3–5 HRS.	10–12 HRS.	17–18 HRS.	21–27 HRS.	42 HRS.
	1926										
6	12–31	0	18.2	38.5	46.8	32.0					
	1927										
7	1–13	0	0	0	9.09						
8	1–24	—	—	—	—	—	—	—	0		
9	1–24	0	13.83	39.3	50.36	60.7	47.5	—	0	0	0
10	1–27	15.75*	15.17	17.15	15.22†	48.32	61.17	—	—	75.0	
11	2–3	0	—	20.22	35.2						
12	2–18	—	—	—							
13	2–18	0	0	29.0	—	—	35.0				
14	3–24	0	—	32.5‡	50.2						
15	3–26	0	—	—	—	55.8					
16	4–6	—	—	—	—	—	—	23.6§	—	7.9	
17	5–16	0	—	—	3.1						
Average		1.75	9.44	25.24	29.99	49.2	47.89	23.6		41.4	

*Twenty grams of glucose injected.
†Udder inflated.
‡Six ounces of glucose injected.
§Just after second inflation.
—No material available at period indicated.
0Test made, with negative results.

An increased amount of ammonia in the urine may also be due to a condition of acidosis in the body or to derangement of function of the liver. These results, in connection with the increased total non-protein nitrogen and urea in the blood, first noted by Hayden,[15] are suggestive of an upset of the nitrogenous balance of the body, which, considered with a hyperglycemia, point strongly to defective oxidation as an important factor. Gergens and Baumann[20] state that guanidin is very resistant to oxidation in the animal body. If it can be demonstrated than an excess of

guanidin is actually present in milk fever, it may assist in explaining the prolonged nitrogenous disturbance in the blood.

With a hypocalcemia demonstrated and the possibility of guanidin intoxication accounting for some of the symptoms of milk fever, the evidence incriminating the parathyroid glands is strengthened. Their regulation of the calcium metabolism of the body is now generally accepted; their regulation of the guanidin metabolism is not so clear and is held in doubt by some. At this stage, although some of the symptoms of milk fever point to the presence of guanidin, it is a necessary requirement that its presence, in the body in undue amount, shall be actually demonstrated.

The more refined technic, in recent years, for blood analysis has made it possible to extend and simplify our knowledge of certain of the constituents. This has been of great benefit in the diagnosis and prognosis of certain diseases and is demonstrating its value in connection with milk fever. In conjunction with urine analysis, information of value has already been gained and there is promise of still greater achievement and ultimate solution of the problem. The inflation of the udder, so effective as a cure and so simple of application, may be found to produce more manifold effects than are generally attributed to it.

The findings suggest as a remedy for the conditions found, in the way of prevention, that a diet containing a sufficient supply of calcium should be available during the period of pregnancy; and for a time prior to parturition and for a short time after, that a limitation of the nitrogenous food would be desirable. Little and Wright, under experimental treatment, state that benefit has been derived from both intravenous and intramuscular injections of calcium chlorid in 10 per cent solution, 30 to 40 cc being injected and repeated, if required, in six to eight hours. When possible, calcium lactate can be given orally in doses of 40 grains every three hours with benefit. Udder inflation in conjunction with the above treatment is an advantage.

Summary

1. The cases of milk fever (44) examined in our laboratory have failed to show a true hypoglycemia, but rather an increased amount of blood sugar, agreeing in this respect with the recent results of some other investigators.

2. The average (75 tests) for blood glucose of 51.75 mg. per 100 cc of blood, established by Hadyen a few years ago, appears

to be none too low. Later tests on cows, for a period of ten monthly intervals, indicate a still lower average of 45.5 mg. per 100 cc (48 tests). A range of 40 to 60 mg. is apparently a safe one for the average normal cow. Hayden's averages for other blood constituents (total non-protein nitrogen, urea, creatinin, uric acids and chlorids) may serve as standards for comparison with pathological conditions.

3. No important difference in the amount of the blood-sugar content in dry cows and those in milk has been observed in our experiments.

4. The fermentation test applied to the blood of milk-fever patients has demonstrated in our cases, with only one exception, that the hyperglycemia at the onset of the disease is due to glucose and that lactose generally did not appear in the blood until after inflation of the udder. Following inflation, the moderate increase in glucose in the blood possibly is due to its non-conversion into lactose in the mammary gland.

5. The experimental injection of 3½ to 4 liters of normal saline solution into a normal cow, aside from its effects on the circulation, produced a stimulating effect upon the kidneys and intestines and quite markedly increased the glucose content of the blood.

6. The experiments of Seitter afford evidence that inflation of the udder increases the blood-pressure.

7. Hayden's examinations of the blood of milk-fever cases reveal an increase in the non-protein nitrogen constituents and indicate that the nitrogenous metabolism is not restored to normal until after the "apparent" recovery of the animal.

8. A marked increase in the amount of the urinary ammonia may assist in the explanation of a hyperglycemia in milk fever. The experiments of Horvath are suggestive; but an endocrine factor may be involved. Watanabe's experiments with guanidin hydrochlorid suggest its possible connection with the increased amount of urinary ammonia, which may also be correlated with a condition of acidosis in the body or a derangement of hepatic function.

9. The hyperglycemia and disturbed nitrogenous metabolism point to defective oxidation processes in the body.

10. The deficient amount of calcium in the blood, with a possible increased amount of guanidin in the body, affords strong evidence that the parathyroid glands are involved.

11. The various conditions referred to, with more or less supporting evidence, indicate the presence of a complex series of phenomena in milk fever.

Acknowledgments

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References

[1]Fish, P. A.: The physiology of milk fever. I. Corn. Vet., xvii (1927), 2, pp. 99-120.
[2]Widmark, E., & Carlens, O.: Parturient paresis: A hypoglycemic coma. No. Amer. Vet., vi (1925), p. 28. (Translated by C. A. Nelson.)
[3]Auger, L.: Recherches sur la pathogénie de la fièvre vitulaire. Rev. Gén. de Med. Vét., xxv (1926), p. 353.
[4]Hayden, C. E.: The blood sugar of the cow in milk fever. Corn. Vet., xvii (1927), pp. 121-131.
[5]Moussu, G., & Moussu, R.: Glycemie normal chez les bovides. Glycemie et maladie vitulaire chez les vaches laitieres. Compt. Rend. Acad. Sci., cxxxii (1926), 7, pp. 431-433. Abst. in Jour. A. V. M. A., lxxi (1927), n. s. 24 (1), pp. 89-90.
[6]Hayden, C. E., & Sholl, L. B.: The blood and urine of the cow in milk fever. Rpt. N. Y. State Vet. Coll., 1923-24, pp. 91-101.
[7]Kaufmann & Magne: Sur la consommation du glucose du sang par le tissu de la gland mammaire. Compt. Rend. Acad. Sci., 1906, p. 779.
[8]Seitter, Max: Experimentelle Untersuchungen über die physiologische Wirkung der Luftinsufflation in das Euter von Kühen und Ziegen als Beitrag zur Aetiologie der Gebärparese des Rindes. Inaug. Dissertation, Stuttgart, 1910.
[9]Little, W. L., & Wright, N. C.: A review of some modern theories of milk fever. Vet. Jour., lxxxii (1926), 4.
[10]Healy, D. J., & Kastle, J. H.: Parturient paresis (milk fever) and eclampsia. The toxic character of the colostrum in parturient paresis. The internal secretion of the mammae as a factor in the onset of labor. (Three papers.) Ky. Agr. Exp. Sta. Bul. 160 (1912).
[11]Horvath, A. A.: Ammonia and blood sugar. Jour. Biol. Chem., lxx (1926) pp. 289-296.
[12]Halliburton, W. D.: Handbook of Physiology (17th ed.; 1924), p. 331.
[13]Paton & Orr: Veterinary Physiology (3rd ed.; 1921), p. 209.
[14]Dryerre, H., & Greig, J. R.: Milk fever: Its possible association with derangements in the internal secretions. Vet. Rec., v (1925), pp. 225-231.
[15]Hayden, C. E.: The blood and urine of the cow in milk fever. (Second report.) Rpt. N. Y. State Vet. Coll., 1924-25, pp. 200-205.
[16]Little, W. L., & Wright, N. C.: The etiology of milk fever in cattle. British Jour. Exp. Path., vi (1925), pp. 129-134.
[17]Cameron, A. T.: The biochemistry of calcium. II. The practical application of our present knowledge of calcium metabolism. Canadian Med. Asso. Jour., xvi (1926), pp. 753-764.
[18]McCollum & Simmonds: Food, Nutrition and Health, p. 32.
[19]Watanabe, C. K.: Studies in the metabolic changes induced by administration of guanidine base. II. The influence of guanidine upon urinary ammonia and acid secretion. III. The relation between the tetanoid symptoms of guanidine administration and the condition of acidosis. IV. The influence of the administration of calcium upon the blood sugar content in rabbits with guanidine hypoglycemia. Jour. Biol. Chem., xxxiv (1918), pp. 51, 65, 73.
[20]Gergens & Baumann: Pflüger's Arch., xii (1876), p. 205.
[21]Paton, D. N. et al: The parathyroids etc. Quar. Jour. Exp. Physiol., x (1916-1917), p. 203. (An extensive series of papers in which are included observations upon guanidin.)

Discussion

Dr. Roger S. Amadon: I would like to present some analyses we have made at the School of Veterinary Medicine, University of Pennsylvania, upon blood samples secured from normal animals; also from a limited number of milk-fever cases, and some samples we have obtained from animals before and after the milking operation.

We have examined thirty-six normal single samples and we have found that the blood sugar ranged from 40 to 86.9 milligrams per 100 cc, 68.5 being the normal average. Our normal milking samples were secured from only four cows, and we found that the variation was so slight that it was practically nil. The drawing of the milk apparently does not upset the sugar concentration in

the blood. Our milk-fever samples have been rather limited in number. We have had to depend upon the practitioner for securing these samples and of course he is limited as to time, and, consequently, we have been rather unsuccessful in getting any great number of these samples into the laboratory. Our seven milk-fever samples range from 55.5 to 100 milligrams, with an average of 72.8. After determining the normal concentration of sugar and noting the concentration in these milk-fever samples, we thought it advisable to find out just what concentration of sugar exists in the blood of an animal upon the development of the comatose condition and other symptoms of milk fever, so we secured a sample of insulin from the Eli Lilly Company, and I will present to you the details of an experiment which we carried out upon one animal. This animal was a cow that had been brought to the Veterinary School for use in the Department of Anatomy. On May 13, at 2:35 p. m., we secured a blood sample, and the animal was immediately given an intravenous injection of insulin, 480 physiological units being administered. The pre-injection sampe taken at 2:35 read 54 milligrams of sugar. One hour later, a second sample showed a concentration of 47.6 milligrams. One hour later, the blood-sugar level had dropped to 43.4 milligrams and the collection of samples was discontinued for the afternoon.

The following morning, at 10:05, we secured a pre-injection sample which read 55.5, showing that a recovery had taken place during that period of time, and we again injected 480 units of insulin and took the following samples during the course of the forenoon. At 11:05 we took the first post-injection samples, which read 54 milligrams; at 12:05 the sugar level had dropped to 41.6; at 1:50 the sugar level had dropped to 37, and at this point we made another injection of insulin to insure a thorough reduction of the sugar concentration in the blood and injected 800 units. We continued taking samples, and at 2:50 the blood-sugar level was down to 33.3; at 3:50 it dropped to 30.3; and at 4:50 it dropped to 23.5.

Fifteen minutes after the drawing of this last sample, the animal was found lying on the right side with the head resting against the left thoracic wall. She took no notice of the attendant who went into the stall to ascertain whether or not she was alive.

At 7:00 p. m., she was still recumbent, but more alert and apparently recovering somewhat from the cerebral depression.

We obtained a blood sample and immediately after the withdrawing of this sample, 200 grams of glucose in a liter of physiological salt solution was given intravenously. The blood sample at 7:00 p. m. still read 23.5 and the sample taken immediately after the injection showed that the concentration had jumped to 250 milligrams per 100 cc of blood, which shows that a large injection of glucose is unessential for bringing the blood concentration up to its level of approximately 68.5 milligrams per 100 cc.

This, of course, is but one case, and we would probably find a variation in animals as regards the reaction to hypoglycemic conditions; we would probably find that some would develop these comatose symptoms at different levels. It does indicate, however, that the concentration that we find in milk fever is far above the concentration that accompanies the train of symptoms that we know as milk-fever symptoms—the cerebral depression and the fall in body temperature.

I believe, Dr. Fish, your lowest concentration in milk fever was 45, and our lowest reading in milk-fever samples was 55.5, which is far above the normal concentration; this is far above the concentration where these symptoms appear.

I do not think there is any question but that the hypoglycemic theory fails to apply to the majority of milk-fever cases. We occasionally encounter the milk-fever case that will not respond to the inflation treatment; we may possibly find in those cases some derangement of the sugar content, and such cases might be benefitted by glucose injections. I believe further investigation of the calcium content of the blood is going to give us additional data and probably will be a more correct explanation of why these nervous symptoms appear in milk-fever cases.

HOG CHOLERA IN THE YOUNG PIG*

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In the susceptible pregnant sow the protection of the young pig against cholera frequently becomes a practitioner's problem before farrowing time and remains so until the young offspring can be given a solid and lasting immunity against the disease. Abortion may follow vaccination by the simultaneous method and even though abortion does not occur, the vaccination may in some instances interfere with the normal development and endanger the lives of the fetuses, resulting in part or all of the litter being expelled dead at full term of pregnancy, a condition commonly called "still birth."

Pregnant sows are often presented for treatment when cholera is imminent. The owner wishes to know how he can most economically protect his animal and the pigs she is carrying. The thrift and life of the sow is the first consideration and the thrift and lives of the pigs she is carrying are of secondary importance. The cost of satisfactorily protecting a sow for any great length of time with serum alone is prohibitive in most cases. If the serum-alone method is used, full-sized doses of serum must be given at three- or four-week intervals. Therefore, the simultaneous method is the one usually preferred.

In administering serum and virus simultaneously to a pregnant sow, two of the common rules of vaccination are of outstanding importance. One is the gentle but firm handling of the animal when she is confined for treatment and the other is good protection with serum against the virus used. Close quarters for catching, such as a small pen, a narrow passage-way or a corner with a hurdle across it furnishes an approach. Obstacles that may be jumped upon or over and slippery surfaces, such as wet cement floors or mud, should be avoided.

A rope noose placed in the mouth and drawn tightly over the nose, or a type of holder which can be similarly applied and either fastened to a solid object or held, is a simple and fairly safe way of confining the animal while she is being injected.

In regulating the dose of serum to be given with the virus, the aim should be to give enough to prevent the slightest thermal or

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other clinically noticeable reaction. Either pregnant sows, or those suckling pigs, require a great deal of serum protection. The writer has recently given 500- and 600-pound healthy pregnant sows in a cholera-infected herd as much as 140 cc of bloody serum with 2 cc of virus. This was perhaps at least a 40-cc margin of safety. The liberal dosage was used for self-explanatory reasons: (1) infection in the herd; (2) pregnancy; (3) the sows were heavy and valuable; (4) the owner was skeptical of the outcome and would have been a poor loser had abortion, sickness or death occurred after vaccination. The results were good enough to indicate that the same doses on the animals would have been justifiable had they been in a healthy herd instead of a diseased one.

All still births following the simultaneous treatment of pregnant sows are likely to be charged to the vaccination, but anyone who has closely observed swine reproduction for a number of years knows that still births are very common in apparently healthy sows that have not been vaccinated or exposed to hog cholera virus in any way.

The owner who wishes to blame the vaccination of his pregnant sows for all of the pigs expelled dead at full term of pregnancy may be reminded of the foregoing fact. The true cause of the large number of still births in swine presents another one of the numerous problems connected with the young pig which needs further investigation.

In the immune sow, the protection of the offspring does not become a practitioner's problem so early; in fact, not until some time after birth. Experimentally, however, the young pig's existence in utero is of interest because of the remarkable resistance to virus which he shows for some time after being farrowed by his immune mother.

Baby-Pig Vaccination

Reynolds[1] reported, in 1910, that young pigs from immune mothers were found to have a high grade of immunity, while those from susceptible sows were found to have practically no resistance to cholera. The same investigator proved that the immunity in young pigs from immune mothers could be made permanent by administering virus alone. Regarding the cause for the presence of immunity in young pigs from immune mothers, Reynolds[2] observed in 1911 that the work so far as he had carried it would indicate that nursing had little to do with the pig's

immunity but he made no study or distinction regarding colostrum. This phenomenon is important because it is closely linked with measures of prevention which the practitioner should take when called upon to deal with pigs of this class.

I wish to describe, as briefly as possible, one experiment that was performed for the purpose of locating the source of the remarkable passive immunity shown by an immune sow's pigs. Thirty hours after birth, six pigs from a long-time immune sow and five from a susceptible sow were exchanged. Three weeks later, four of the six pigs from the immune mother, suckling the susceptible sow, were each given 1 cc of virus alone. Two pigs were left as controls. Five days after the injection of virus, the four pigs showed roughened coats and a very slight droopiness which in four days disappeared. The controls showed no indisposition throughout the test.

At the same time three of the five pigs farrowed by the susceptible sow and suckling the immune sow were each given 1 cc of the same virus with two of the pigs left as controls. Five days later one of the injected pigs was slightly droopy and six days after the injection the litter had the appearance of a serum test with the three injected pigs lying sick and hidden in the nest while the controls were vigorous and active.

A few days later the two controls in this second litter contracted cholera by contact and in seventeen days after the injection of virus alone the entire litter was dead of cholera.

The immune sow had been injected with virus alone some time before breeding and therefore had been proved immune. But the value of the experiment rested upon whether the supposedly susceptible sow was truly susceptible.

After the test was over and the susceptible sow's adopted pigs were eight weeks of age, they were weaned and she was given 2 cc of virus. The fifth day after injection she had a temperature of 107.4° F. She died on the eighth day. The diagnosis after autopsy was acute hog cholera.

It was evident from this experiment that the pigs which were thirty hours old when taken from their immune mother had a grade of immunity sufficiently high to withstand the injection of 1 cc of potent virus after they had suckled a susceptible sow for three weeks, while the pigs farrowed by the susceptible sow that were likewise taken from their own mother when thirty hours old and had suckled an immune sow for three weeks, showed no resistance to the virus.

It appears that the prenatal influence of the immune sow is the big factor responsible for the phenomenon of passive immunity against cholera in her young pigs. However, the fact that both of the two litters involved in the experiment suckled their own mothers for thirty hours must not be lost sight of and in the light of present knowledge, the colostrum of the immune mother must be considered a possible medium through which antibodies may be transmitted.

Another fact that must not be lost sight of is that the decisiveness of this experiment could not be expected regularly. It has been found in other experiments that pigs farrowed by and suckling their own immune mothers will frequently not withstand 1 cc of virus alone at three weeks of age. For example, each of a litter of seven pigs farrowed by and suckling an immune sow was injected with 1 cc of virus alone. In fifteen days after the injection, three of the seven had died of cholera. The diagnosis was confirmed by injecting 3 cc of the heart-blood of one of the pigs into a susceptible shote.

On the other hand, it has been found that pigs from immune mothers may show a high grade of immunity at an age more advanced. As an example of this, a test was run on a pig nine weeks old from an immune sow and two pigs of about the same age and weight from a susceptible sow. Each of the pigs was given 5 cc of virus. The two pigs from the susceptible sow were sick enough with cholera to kill for virus on the fifth day after the injection, while the pig from the immune sow showed a high temperature twenty-four and forty-eight hours after the injection, due mostly to hot weather and excitement, and other than that showed no clinical changes.

The litter mates of the immune pig in the foregoing test were tested at twelve weeks and one day of age with a susceptible pig of about the same age and size. Each of the three pigs was injected with 5 cc of virus. The pig from the susceptible sow was sick enough with cholera to kill for virus on the fourth day after injection and the two pigs from the immune sow showed a temperature reaction, slight droopiness and partial loss of appetite but soon returned to normal.

Natural Immunity to Cholera

Hoskins,[3] in 1915, reported that the previous year, in routine serum-production, out of 1628 pigs weighing from fifty to one hundred pounds he found 232, or 14.2 per cent, either partially

or entirely resistant to the injection of 2 cc of potent virus. The possibility of some of these pigs having been previously vaccinated is mentioned by Hoskins and the disadvantages to the serum-producer of using these highly resistant pigs in serum tests are clearly described. It is evident that if a young pig with an unknown history is injected with a highly potent virus, as is done in serum plants in the propagation of virus, either in or out of the serum test, and shows no reaction, it is impossible to say whether the immunity observed is due to the vaccination of the pig itself or to the fact that he had an immune mother.

Enough evidence of this nature is at hand to show very plainly that we have a phenomenon in the young pig farrowed by the immune sow which is of sufficient importance to receive the careful attention and consideration of not only the practitioner and serum-producer but the swine-grower as well.

It is an advantage to the practitioner to keep in mind that pigs from immune mothers show varying grades of passive immunity which will frequently withstand 1 cc of virus alone at three weeks of age and in some instances is sufficient to withstand 5 cc of virus alone at nine weeks of age; that the source of this immunity is either prenatal or from the colostrum and does not depend on suckling the immune mother but is coincident with it. Also, no matter how high this grade of immunity is in the young pig, it disappears and after a certain age, varying in individuals even within the same litter, it disappears rather rapidly to a point where either natural or artificial exposure to cholera virus may result in sickness and recovery or sickness and death.

The question confronting the practitioner is in regard to the time when he can most economically convert this passive immunity into an active one by administering serum and virus simultaneously. This question I wish to discuss later.

Passive Immunity a Problem

The serum-producer's problem is how he may avoid this passive immunity in his serum tests and in pigs which he wishes to use in virus production. If a pig from an immune sow is injected with virus alone when his maternal immunity, as it may be called, is waning, and he becomes sick with a more or less chronic type of cholera and shows good lesions of the disease upon autopsy, will his virus when used in the simultaneous treatment produce a solid and lasting active immunity and if used for hyperimmunizing will it produce a hyperimmune that will in turn produce serum of

high potency? This question is very important in connection with serum and virus production but cannot be discussed in this paper.

The swine-grower's interest in the fact is that if he maintains an immune herd the active immunity which is conferred upon one crop of pigs by the proper administration of potent serum and virus is continued in the offspring in the form of a passive immunity for varying lengths of time after birth. In protecting the second generation, it will save him money if his veterinarian can add enough serum protection to the passive immunity already present by injecting with the virus used in the simultaneous treatment enough anti-hog cholera serum to confer upon the young pig a solid and lasting immunity.

The resistance present, also the small size of the pig, very materially cuts the cost on serum if the active immunity can be conferred at such a time rather than wait until the pig has increased in body weight and until his maternal resistance to cholera has disappeared so that he must get protection entirely from the serum injected.

Before discussing immunity further, I wish to mention another phenomenon observed in the young pig which may, sooner or later, puzzle one who is called upon to examine him. This phenomenon is connected with the diagnosis of cholera in the very young pig. Up to about three days of age the losses from pigs expelled dead, from chilling immediately after birth, from insufficient nourishment and from being stepped upon and lain upon by the mother are sometimes very great. In holding autopsy regularly on these very young pigs I find that most of them show lesions which one would expect to find if they had been infected with cholera virus. I have found in a large majority of these very young pigs kidneys that were peppered with petechiae. A few show petechiae on the mucous lining of the bladder and a few show petechiae on the heart and lungs. The petechiae on the kidneys are especially suggestive of hog cholera. These have been found in pigs from susceptible sows as well as pigs from immune sows but most of the pigs examined were from immune sows. Upon injecting heart-blood and other tissues into susceptible shotes, hog cholera was found to be absent. Clinical symptoms in the very young pig are not of a distinctive nature. Therefore, unless one can confirm a diagnosis by injecting a susceptible pig with heart-blood, it is difficult, if not impossible, to make a diagnosis. Some time has been given to studying the significance of the

petechiae in the kidneys of very young pigs but no conclusions have been reached and I have been unable to find in literature anything concerning them.

The most important phase of cholera in the young pig is from the standpoint of immunology. The periodical hog cholera wave which has only recently caused very heavy losses to swine-growers, emphasizes again the importance of immunizing against the disease.

Lack of definite knowledge of the young pig and his reactions to anti-hog cholera serum and cholera virus have made practicing veterinarians uncertain as to what method of vaccination to recommend to swine-growers. Such an attitude on the part of the veterinarian who is the farmer's authority on such matters, as one might reasonably expect, has made the farmer uncertain and hesitant.

Vaccinating swine of advanced age is expensive when compared with the cost of vaccinating young pigs. High cost of vaccination is an important retarding influence in the control of hog cholera. Again, the large number of so-called "vaccination breaks" in 1926 raised the question whether pigs vaccinated at an early age that died later with cholera were treated too young.

For the past twenty years vaccination against hog cholera has been known as a sure preventive of the disease. The work which Dr. Dorset and his associates have done on hog cholera virus cannot be questioned. It ranks in importance with Pasteur's work on rabies, with Koch's on tuberculosis and Smith's on Texas fever. But the application of the method is far from perfection. We may well give serious consideration to present conditions.

Farmer Has Paid for Mistakes

Some mistakes have been made in research, in serum and virus production and in the field. For a number of years the farmer who has had vaccinating done has paid for these mistakes. There has not been sufficient research since Dr. Dorset's great achievement. The practitioner has been compelled to use what meager information he could get, regardless of the source, and learn the rest by his own experience in the field at the farmer's expense. Some of the research work on hog cholera has been done on the "American plan," consisting of short superficial studies with overdrawn and over-emphasized conclusions. The serum-producer has too frequently taken a chance by sending

out products with questionable potency and the farmer has paid. The practitioner has at times made too much profit on serum and virus used and has charged too much for his services. He has too frequently done his work hurriedly and carelessly, giving the farmer the impression that no special knowledge or technic is necessary. When called upon to investigate a vaccination break he has too frequently failed to make an honest effort to find the true source of the trouble and place the responsibility where it belongs. In such cases he has frequently deserted his farmer client entirely and has protected himself and the producer of the serum and virus. Now he is wondering why the farmer is deserting him.

Furthermore, the practitioner has too often allowed commercially made swine diseases with commercially made preventives and cures to cut his clients' profits on pork production. Last, but not least, we have sometimes failed to advocate firmly and impressively the cheapest and safest method of preventing cholera. The farmer, in an effort to protect himself and cut at least a part off of his vaccination bill, is now attempting to do his own vaccinating and it remains to be seen just how well he will succeed. Then, too, farmer vaccination is being promoted by the farmer's efficiency engineer, the county agent, and while it seems to us like a short-sighted program, we cannot be sure of the final outcome. It seems a short-sighted program because it may in time mean that the farmers who are doing their own vaccinating, as well as others in the same locality, will be deprived of all veterinary service. Doing the hog cholera work and not doing it makes the difference between a good practice and a poor one to many practitioners in the Middle West. Conditions of this kind do not induce good men to stay in or to enter the profession.

More Study Needed

In the meantime we should do everything possible to correct mistakes of the past, hoping that the farmer will soon see the folly of attempting to do work for which he is not qualified. With our present knowledge, vaccination of the young pig furnishes us the cheapest and safest way of protecting the swine herd against cholera. This statement is simple. But when we attempt to speak in definite terms and say just how and when the young pig should be vaccinated in order that he may have a solid and lasting immunity we invite argument.

The controversy involves two very perplexing agents, i. e., hog cholera virus and the young pig. They seem to act and react in capricious ways. However, after we learn more about the true nature of hog cholera virus and the young pig we will without a doubt find definite laws governing them. Before one can be sure of such laws they must be observed many times by ourselves and others. In the case in hand, we find a wide difference in results and in the interpretations of results observed by various investigators. Each one conscientiously adds his bit and in the final reckoning certain facts become established.

It is extremely difficult, if not impossible, for the busy practitioner to give all evidence on both sides of a question the time and thought necessary to decide which side is the right one. But while some men are devoting their time attempting to solve the puzzling problem, the veterinarian in the field must continue to use a method of some kind even though it may not be the one most advantageous to his client. He must continue to vaccinate pigs of various ages, despite the fact that the question of just how and when to vaccinate them has not been settled.

It is also extremely difficult, if not impossible, for one observer to pass judgment and advocate one method over another without seeming dogmatic or being accused of having a selfish motive. Realizing fully the delicate situation, I can only present my opinions to you and let you judge whether they be right or wrong. My only motive for doing this is to help the swine industry through the medium of the veterinarian.

Three Methods Employed

The history of the problem is short. There have been but few changes or modifications attempted since the simultaneous treatment was originated. In 1912, Schoenleber,[4] of Kansas, issued his bulletin (No. 182) in which he described three methods of vaccinating: (1) the serum-alone; (2) the simultaneous; (3) the combination method. This combination method is the one which Schoenleber also called the double treatment and the same that Hoskins, in 1915, termed the "follow-up method."

Schoenleber writes of his third or combination method:

> The third method is the one which was originated and thoroughly tested by the writer and is a combination of the first and second methods. Vaccinate first with the serum alone, and ten days later vaccinate with the simultaneous method. This is by far the most satisfactory and safe method in an exposed herd, or if there is cholera in the herd or in the vicinity; or if the hogs are fat or otherwise valuable.

I had been a student in Dr. Schoenleber's department just previous to the time this bulletin was issued and we had been informed by him of the complaints of serum breaks that were coming to his office. The origin of the combination treatment was an attempt to stop the stunting effect, sickness and death that sometimes followed the simultaneous treatment and I can testify to the fact that it was a very safe method for, if you will pardon these personal references, I will say that in starting into practice, in 1911, in the corn and hog section of eastern Kansas, I used Schoenleber's combination or follow-up method on all hogs, regardless of age. It was as the originator said, safe but expensive, and it was soon discontinued on account of the heavy expense.

Dr. Schoenleber found that by increasing the dose of serum in the simultaneous method he could eliminate the harmful effects which had caused so much complaint when smaller doses were used.

No changes were again attempted until 1918, when Cahill[5] and Birch,[6] working separately, started to advocate a follow-up treatment for baby pigs only. The follow-up method this time consisted of giving a serum-alone treatment at six weeks of age and following it at twelve weeks of age with the simultaneous treatment. This was advocated as a method particularly suitable for hogs in the East, in contradistinction to those in the Middle West. The object of this new follow-up method was to allow pigs that were candidates for the simultaneous treatment to become nine, or preferably twelve, weeks of age before administering the simultaneous treatment in order that they might receive a solid and lasting immunity. Cahill and Birch also wished to avoid a stunting effect on young pigs which seemed to follow the simultaneous treatment if given without the preceding treatment of serum alone.

Follow-up Method Discontinued

The writer began to vaccinate hogs in the East in 1921. Cahill's and Birch's sectional discrimination was heeded and their follow-up method adopted. It was used for a time but I gradually returned to the simultaneous method. The change back has been made cautiously but almost completely. So far as the writer knows, no one has proved that the follow-up method of vaccinating young pigs has any advantage over the simultaneous method when serum and virus of high potency are used

in the proper manner and quantities. As far as is shown, Cahill thought he used a virus of at least standard immunizing properties in his experiment when he vaccinated the 852 pigs weighing fifteen to thirty pounds. It is the opinion of the writer that he unknowingly and of course unintentionally, used a low grade of virus which still had potency enough to produce sickness and finally death when injected into a susceptible pig but not the high grade of potency that is required to produce a solid and lasting immunity.

It is the writer's opinion that the vaccination break in Cahill's experiment was not due to the early age of the pigs vaccinated but to the low grade of virus used and possibly to a low grade of serum also. I say possibly to a low grade of serum because this point depends largely on how soon after vaccination the fifty-two pigs, not accounted for by Cahill, died.

Cahill got exactly the kind of results that the writer from his own experience would expect to get from a low-grade virus. He got an increasing percentage loss after each test injection of virus alone. At eight weeks he lost 21 per cent; at twelve weeks, 48 per cent; at sixteen weeks, 52 per cent; at twenty weeks, 64 per cent; at twenty-four weeks, 72 per cent; which shows that as time went on the low-grade virus immunity was waning. We may finally learn that it takes a sharper virus in the very young pig, under fifteen pounds in weight, to produce a solid and lasting immunity than in the pig weighing from fifteen to thirty pounds. But with our present knowledge I am far from convinced that fifteen- to thirty-pound pigs, vaccinated with 30 cc of potent serum and 2 cc of potent virus, will be susceptible in eight, twelve, sixteen, twenty and twenty-four weeks to the extent of 21, 48, 52, 64 and 72 per cent when given 4 cc of virus alone, as was the case in Cahill's experiment. It is seldom necessary to vaccinate pigs under fifteen pounds except in susceptible herds threatened with exposure. I personally prefer to vaccinate pigs of the weight mentioned by Cahill (15 to 30 pounds) and so far have observed no evidence of an endogenous condition preventing or hindering the formation of a solid and lasting immunity following the administration of the simultaneous method when using highly potent serum and virus.

Serum-Alone Protection Sometimes Short

Again, Cahill justifies the six-week interval between his serum-alone treatment and simultaneous treatment by saying: "Passive

immunity conferred by serum-only treatment in the East seldom or never fails to last for six weeks." I disagree with Cahill on this point. I have seen serum-alone protection broken through by cholera in less than three weeks in both the East and Middle West. According to Porter's[7] work, a serum-alone break under three weeks is the exception rather than the rule but these exceptions occur too frequently to be ignored. Very little if any difference has been observed by the writer in hog cholera and hog cholera immunology in the East and Middle West. I consider the six-week interval, as advocated in the follow-up method, unsafe.

Birch shows no experimental data that indicate a superiority of the follow-up method over the simultaneous method. He lays considerable stress on clinical experience but the evidence I have causes me to believe that he also misinterpreted results.

Since Cahill published his startling results and both Cahill and Birch began advocating the follow-up method, this phase of hog cholera immunology has been reported upon by Niles and Rietz,[8] Pickens, Welsh and Poelma,[9] Dorset and Buckley,[10] Graham, Boughton and Tunnicliff,[11] Benner [12] and perhaps others. In these experiments there are important exceptions to a solid and lasting immunity being produced after the simultaneous method but I have also seen such exceptions in the field where the follow-up method was used. It is the opinion of the writer that these exceptions will not be eliminated until the virus pig and the virus receive much closer attention than they have in the past.

Conclusions

In conclusion, I wish to say that the principal points in attempting to protect the young pig against the effects of cholera virus before birth are the careful handling of the sow and good serum protection. The young pig from the long-time immune sow shows a variable resistance to cholera which is an asset in vaccination by the simultaneous method but a liability in serum tests and virus production. One experiment reported here indicates that this resistance is transmitted from mother to offspring, either in utero or through the medium of the colostrum.

In holding autopsy on pigs under three weeks and even up to five weeks of age, one frequently finds petechiae on the internal organs which are not necessarily indicative of cholera.

The earlier use of the simultaneous method is marked by Schoenleber's attempt to prevent stunting effects, sickness and

death following the simultaneous treatment. To do this the combination method was originated which was safe but too expensive. When it was later discovered that the same results could be obtained by increasing the dosage of serum, the combination method was discontinued. The later use of the simultaneous method is marked by Cahill's and Birch's attempts to avoid the inability which the baby pig was thought to have of forming a permanent immunity when receiving the simultaneous treatment and to prevent the stunting effect which followed its use. In order to do this another follow-up method was originated and advocated which is like Schoenleber's in that it is too expensive, but in addition is unsafe. The writer believes this latter method was based on a wrong interpretation of the results of an experiment by Cahill and a mistaken idea of the cause of harmful effects seen in the field by Birch.

Furthermore, it is the writer's opinion, based on experience in the Middle West and East, as well as the experience of others, that the simultaneous treatment confers a solid and lasting immunity to pigs weighing fifteen pounds or more, and that this can be done without harmful effects when a potent virus is used with ample doses of potent serum.

REFERENCES

[1]Reynolds, M. H.: Immunity in young pigs from cholera immune sows. Amer. Vet. Rev., xxxviii (1910), pp. 236-237.
[2]Reynolds, M. H.: Hog cholera. Amer. Vet. Rev., xl (1912), pp. 485-492.
[3]Hoskins, H. P. Some observations on hog cholera and the use of serum. Amer. Vet. Rev., xlvii (1915), pp. 46-56.
[4]Schoenleber, F. S.: Vaccination against hog cholera. Kans. State Agr. Exp. Sta. Bul. 182 (1912).
[5]Cahill, E. A.: Hog cholera control in the East. Jour. A. V. M. A., liv (1919), n. s. 7 (4), pp. 314-321.
[6]Birch, R. R.: Observations in regard to immunising young pigs. Corn. Vet., ix (1919), 2, p. 75.
[7]Porter, E. W.: Some experimental work in hog cholera. Jour. A. V. M. A., lxiii (1923), n. s. 16 (5), pp. 580-590.
[8]Niles, W. B., & Rietz, J. H.: Duration of immunity against hog cholera following simultaneous inoculation of young pigs. Jour. A. V. M. A., lvii (1920), n. s. 10 (2), pp. 176-182.
[9]Pickens, E. M., Welsh, M. F., & Poelma, L. J.: The susceptibility of young pigs to hog cholera. Jour. A. V. M. A., lviii (1921), n. s. 11 (4), pp. 403-412.
[10]Dorset, M., & Buckley, S. S.: The immunization of young pigs against hog cholera. Proc. 29th Ann. Meet., U. S. Live Stock San. Asso., 1925, pp. 43-52.
[11]Graham, R., Boughton, I. B., & Tunnicliff, E. A.: Observations on the duration of cholera immunity in baby pigs following serum and virus treatment. Jour. A. V. M. A., lxx (1927), n. s. 23 (4), pp. 448-458.
[12]Benner, J. W.: Immunising young pigs against hog cholera. Rpt. N. Y. State Vet. Coll., 1922-23, pp. 204-213; 1923-24, pp. 87-90; 1924-25, pp. 225-230; 1925-26, pp. 106-117.

DISCUSSION

DR. M. DORSET: I have been very much interested in Dr. Benner's paper. He has already indicated to you the problems that are met with in connection with the immunization of young pigs.

The question, as I address myself to it, is: Shall pigs be immunized by the simultaneous method during the suckling period before weaning? It seems to me that you have to consider this question from two aspects: (1) Can you, as a matter of scientific fact, confer a lasting immunity upon a young pig, or are young pigs, for some reason unknown to us, unable to acquire a lasting immunity following simultaneous treatment? (2) The other aspect is that

even, if it is scientifically possible to immunize them, is it desirable, is it advisable, to do it?

The work done in the Bureau of Animal Industry was carried out by Dr. S. S. Buckley, of the Animal Husbandry Division, and myself. Our studies of the immunization began in 1921 and have continued since that time regularly on all pigs farrowed in the spring and fall, at each of the seven government farms which we have had at our disposal. We have regularly immunized the litters at these seven farms spring and fall for an average of more than six years. These farms are located in Maryland, Mississippi, Louisiana, South Dakota and Montana. During this period we have given the simultaneous inoculation to 5695 suckling pigs. It has been the custom to treat all these pigs at about the time farrowing was complete at each of the farms, so that pigs of various ages were treated. The age at time of treatment varied from one day up to ten weeks. Of course we would not advise in practice the treatment of one-day-old pigs. We wanted to know whether a pig could be too young to be immunized. These pigs were all grown for experimental purposes in pig-feeding and pig-management, and we have not been able to test all of these pigs for immunity. We have been able, however, to test the immunity of 1763 of these pigs by injecting them with hog cholera virus after an interval of 2½ to 9 months.

We have tested some pigs from each year's immunization. The results have been essentially that these pigs when tested were always immune except those immunized in the spring of 1924. A large proportion of the 1924 spring pigs were not immune. We came to the conclusion that these pigs in 1924 did not have a lasting immunity on account of the unsuitability of the virus which we had used for immunizing them in the spring of that year. There was good reason to believe that that virus was of reduced potency and I think that Dr. Benner's recommendation of highly virulent virus to get prolonged immunity is a very wise plan indeed. I am perfectly satisfied that the highly virulent virus will not increase the danger to young pigs at all, provided you use enough good serum with it. The object should be always to avoid any symptoms whatever of sickness as a result of immunization.

As a result of these experiments we have come to certain general conclusions. First of all, we believe that age is not a controlling factor, as far as immunization of pigs against hog cholera is concerned. We have regularly found pigs immunized when one week old to be firmly immune when tested several months later.

Second, we have not been able to find that the use of large doses of virus was advantageous. In these experiments we used from 1 to 4 cc of virus, merely to determine whether the use of large amounts of virus would be an advantage. I know some practitioners have regularly administered as much as 5 to 10 cc of virus to every suckling pig. Our experiments indicate that the important thing is to have good virus, and that if the virus is good, one cubic centimeter will be plenty for these suckling pigs.

With respect to the serum dose, we never used in our experiments more than 30 cc per pig. The average was probably about 20. In some cases we used 10 cc of serum and 4 cc of virus. I would not advise that as a general practice. These were pigs from immune sows. A pig from a susceptible sow should be given a more liberal dose of serum.

We do not know why it is that some virus is good and some virus is not so good. That is one of the most important questions before the research men. It has seemed to me that we do not get as good virus in the early spring as we do later in the year. I believe that the collection of virus in the spring is a very important thing for the serum-producer to watch, to make sure that his virus is of the highest virulence.

I do not believe that simultaneous inoculation should be attempted if the suckling pigs are sick. If there is any sign of illness among those pigs, from any other cause but cholera, especially if they have any respiratory affection, I would not give them the simultaneous inoculation. I do not think it makes very much difference which of the two methods Dr. Benner has been discussing —the follow-up method or the simultaneous inoculation—is used, so far as protection against cholera is concerned. That is a question for the veterinarian

and the owner to decide between them. I believe either can be done successfully.

So that, answering the two questions which I propounded, I think from a scientific standpoint there is no question but that a lasting immunity can be conferred upon suckling pigs. Second, answering the other question as to whether it is desirable or not, I should say that it is desirable if properly done and that it is a job for a skilled professional man and not a job for a layman.

DR. I. K. ATHERTON: On listening carefully to Dr. Benner, I am not sure that it was the prospective, foreseeing father that was worrying about the future of those pigs, but I think Dr. Benner was the one that was doing it. He used up his time telling us about the pitfalls that we had to look out for in the use of the double treatment. He told us, however, that it was the only protection we had against hog cholera. Then, my friend, Dr. Dorset, comes along and says that there is a lot we do not know about why virus does or does not have a "kick," and he tells us that under certain conditions we must not use the double treatment. We are told it is the only way we can protect hogs from cholera and yet at the same time we are told we must not use it.

In Maryland we do not use much double treatment except in garbage-feeding plants. In 1925 —remember that there is no one in Maryland who administers the treatment but veterinarians—our "breaks" ran 14 per cent; last year they ran 10 per cent. Mr. Hedgecock, of the Illinois Farm Bureau Serum-Virus Association, told me that they had purchased 35,000,000 or 40,000,000 cc of serum for the farmers of Illinois last year. I will admit that a great deal of this was for layman vaccination, but he told me that in Edgar County, Illinois, he estimated that 60 per cent of the farmers who had the double treatment administered to their hogs suffered losses as a result of the treatment. I was told recently that this large percentage of "breaks" following the administration of the double treatment was unnecessary, and I am going to agree with the man who said it. I think if the double treatment is properly used, there is no place for the single treatment, but I do not know of any biologic that is being abused more today than serum or the combination of serum and virus. This official who told me that we had too large a percentage of outbreaks admitted that if we could reduce those "breaks" to 4 per cent, it would be an almost ideal solution of the hog cholera problem. I did a little figuring for him. I took the outbreaks of cholera which we have had in Maryland for the last eight years, and I showed him that if we administered the double treatment to all the hogs in Maryland each year and had 4 per cent of "breaks," we would have just doubled the number of cases of hog cholera in Maryland over what we did have under what we know as the Maryland Plan.

I do hope that our investigators in the hog cholera problem will not forget to pay more attention to the sources of infection in primary and secondary outbreaks. I believe they will find therein the solution of the hog cholera problem.

DR. R. R. BIRCH: We have had an interesting paper and some interesting and helpful discussions. Progress is made in this way, and sometimes unmade, for often there is a tendency to move from known safe methods to new methods less well proved.

In our Eastern herds, we began, in 1910, the practice now in vogue in the Corn Belt, of vaccinating shoats a few weeks after weaning time. This was usually successful, but in some herds losses began to appear even before weaning time. Eventually these losses became so extensive that we had to change our plan. Others experiencing the same difficulties had been working back toward baby-pig vaccination. I never followed that practice, so you may discount my remarks as much as you wish on that score. I have visited many farms, though, where the men who used it were in trouble, and those experiences have led me to observe caution, perhaps undue caution, in regard to routine baby-pig vaccination.

As a substitute we followed the custom of protecting baby pigs, and older hogs temporarily low in resistance, with a dose of serum alone and following later with simultaneous treatment. This has been an exceedingly safe procedure, but because of its expense we never advocated it as a rountine procedure except for young pigs definitely threatened and other hogs low in resistance.

Dr. Benner is perfectly right in stating that we have no *experimental evidence* to show the superiority of the follow-up treatment over the simultaneous treatment. That is true, but we have *evidence* nevertheless, as all know who have used virus extensively on pigs low in resistance. For instance, a consignment of hogs, after a long journey by rail, reaches its destination. Does anyone with extensive experience want to vaccinate the animals immediately with serum and virus, even though he increases the serum dosage? Does anyone want to give virus to farrowing sows? Yet these animals often require immediate protection. An extra dose of serum alone is often a good investment, and I believe we are right in advising the safer, though the more expensive, plan. Then, if the owner wishes less safe and less expensive work, the choice should be his.

I was glad that Dr. Dorset covered this point, advising against the use of virus on all pigs low in resistance. In our eastern states we encounter many herds which require immediate protection, but which are temporarily low in resistance. We have to give immediate protection, but the pigs, temporarily at least, will not tolerate virus well. Follow-up treatment is the safest method of handling a situation of that kind.

I think perhaps I have been over-cautious in this respect. No doubt I have used follow-up treatment when simultaneous treatment would have answered the purpose just as well and at less expense; but there is good evidence that the opposite course, that of giving simultaneous treatment indiscriminately to all hogs that require protection, even with a little extra serum to those obviously below normal in resistance, would have resulted in much heavier losses in the pigs vaccinated.

Now regarding the experimental work. We hear much about the difference in different viruses. These differences exist and the fact is not to be discounted, but we have also differences in pigs, which are less frequently mentioned. In using the same virus on pigs from the same litter, some acquire permanent immunity as a result, others fail. The failures have appeared in the work of all who have made observations on large numbers of pigs. They are recorded in the work of Dorset, Benner, Cahill, Niles, Tunnicliff and Dimock. Recently Dr. Geiger, of Eystrup, Germany, and Drs. Hutyra and Kokes, at Budapest, informed me that these failures are not uncommon experiences with them. There is thus ample evidence that there exist persistent exceptions to the rule that baby-pig vaccination confers permanent immunity. Until these failures are further explained, or until a like number are shown to occur in pigs vaccinated somewhat later in life, I believe we should use caution in stating that age is not a factor in determining the duration of the immunity conferred by simultaneous treatment.

Drs. Dorset, Benner and Tunnicliff have done a very valuable piece of work in showing that these failures are less frequent than many, among whom I include myself, had supposed them to be. Field trials may now be made without great danger, provided all the pigs are in good condition when vaccinated. Eventually we shall know. Meanwhile, I believe prudent veterinarians will inform their clients that baby-pig vaccination is still on trial, and let them assume the responsibility in initiating it. The experiences of the last two years, which in some quarters have severely shaken confidence in all vaccination, deny the wisdom of offering, without reservations, anything except proved methods.

SENIOR STUDENTS VISIT DETROIT

The senior veterinary class at Michigan State College visited Detroit, the week of April 16, under the guidance of Dr. L. B. Sholl. A visit to the A. V. M. A. headquarters was a part of the itinerary, which included visits to Parke, Davis & Company, the Detroit Creamery stables and farm, packing-plants, veterinary hospitals, etc.

HOG CHOLERA CONTROL AND THE VETERINARIAN*

By M. DORSET, *Washington, D. C.*

Chief, Biochemic Division, U. S. Bureau of Animal Industry

While pondering over the old question of hog cholera control, I found it helpful to consider first of all the methods employed to combat infectious diseases in general, and then, by a process of elimination, to select a plan which seemed suited to the specific case of hog cholera in the United States. I shall therefore ask your indulgence while I review very briefly the general methods of disease control before proceeding to discuss hog cholera in particular.

Infectious diseases of both man and animals are combated either by attacking the causative microorganisms or by building up the defenses of the host.

METHOD OF ATTACKING THE DISEASE-PRODUCING MICROORGANISMS

The methods employed in attacking the microorganisms of disease are varied to suit the particular case but in general they may be placed in three categories:

1. The so-called "slaughter-out method," which has been so strikingly successful in eliminating contagious pleuro-pneumonia and foot-and-mouth disease from the United States. It involves (in such a highly contagious infection as foot-and-mouth disease) immediate destruction of all infected animals and contacts; of all materials, feed and even buildings, which cannot be disinfected; the stoppage of traffic; the closing of markets and the maintenance of the strictest quarantine over a long period of time by an army of inspectors. It is economically possible of application only in limited areas, where the hardships imposed upon the few are justified by the benefits conferred upon the many.

2. The second method of fighting infections may be called the "indirect method." It is based upon the fact that certain diseases are not spread by contact of healthy with infected individuals nor by dissemination of the excretions of the sick, but only, or almost entirely, through the agency of an intermediate host. The transmission of Texas fever by the tick and of malaria

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and yellow fever by certain species of mosquitoes are classical examples of such diseases. In these cases the attack is directed solely against the insect carrier. The extirpation of the Texas fever tick deprives the germ of the disease of the single channel through which it must pass in order to find its way to fresh victims. It is evident that this indirect method is applicable only in those diseases which are transmitted through the agency of an intermediate host.

3. The third method of attacking disease producers may be referred to as the "sanitary police method." This method is generally applied when no better plan can be adopted and is frequently used as an adjunct to more specific and more effective measures. It consists essentially of quarantine of infected and exposed animals and premises; sanitary disposal of the dead and of the excretory products from the living; and cleaning and disinfecting of cars, yards and barns. As a rule this method is applied by the issuance of official regulations that are handed over to a force of inspectors, which, numerically, is wholly inadequate to force compliance. The result is usually disappointing, so that this plan may be regarded as palliative at best, and wholly ineffective to control sweeping epizootics of such highly infective diseases as foot-and-mouth disease and hog cholera.

Methods of Increasing the Resistance of the Host

The maintenance of body vigor by provision of an abundance of adequate food and clean, dry sleeping quarters; by protection against severe weather, particularly cold and dampness; and by the avoidance of exhaustion from overwork, may be regarded not only as essential to good husbandry, but as the basis upon which all specific methods of protection against disease should rest. However, as important as is good hygiene, it alone is not sufficient to enable the animal body to resist invasion by many of the microorganisms to which it may be exposed. To supplement these hygienic measures there have been developed specific means, in the form of vaccines and immune sera, to aid the natural forces of the body to repel the attacks of disease. These biological agents, when available, are applied, usually in combination with the sanitary police method, when the slaughter-out method and the indirect method are impracticable.

Two systems of utilizing vaccines and sera may be recognized:

1. *Wholesale preventive inoculation* We have examples of this system in human practice in the almost universal vaccination

against small pox, in the immunization of school children against diphtheria and in the immunization of all army recruits against typhoid fever. In veterinary practice this system is applied in certain areas to combat anthrax, blackleg, rinderpest and other diseases.

2. In the second system of combating disease by increasing the resistance of the host, the specific anti-sera and vaccines are administered only when disease appears, or when there is imminent danger of infection. The treatment is applied to the infected and to the contacts, and mild sanitary police measures, such as short quarantine of infected premises, cleaning, disinfection and warning notices, are instituted. This system, *if efficiently applied*, may serve largely to eliminate deaths from the disease. Early notification and immediate treatment of the infected herds are essential to success.

Having now reviewed very briefly the various plans which have proved to be of value in the control of animal diseases, we are ready to consider the main topic of this paper, the control of hog cholera, bearing in mind always the economic as well as the *scientific* aspects of the subject.

Hog Cholera Control

In hog cholera we have a disease which is known to have existed in the United States for nearly 100 years, and which, though perhaps most widespread in the Corn Belt, is yet found in every state in the Union. It is highly fatal, extremely contagious and seems to be spread in a great variety of ways, some of which are perhaps not yet recognized. An intermediate host is not necessary for transmission, in fact it has not been proved that insects are ever, in nature, responsible for the spread of the disease. Hog cholera is most prevalent in the late summer and fall and resembles foot-and-mouth disease and human influenza by appearing periodically, after a lapse of years, in the form of a sweeping epizootic. Such great outbreaks of hog cholera have occurred in 1887, 1897, 1912 and 1926. An attack of hog cholera confers a firm immunity upon recovered animals. We have available a serum which will confer a passive immunity upon susceptible hogs; will effect a cure in most cases when administered in the incubative stage of the disease and will confer a lasting immunity when administered simultaneously with the virus of the disease.

With all of the above facts in mind, what method should be employed to control hog cholera losses in the United States? At the very outset, the slaughter-out method may be discarded. Such a system would involve practical destruction of the hog industry and would require a much larger force than the entire veterinary profession could supply. We can likewise immediately put aside the indirect method, since we know of no intermediate host that is responsible for spread of the disease. The sanitary police method is admittedly ineffective, alone, to control such an infection as hog cholera, although, when intelligently applied, it should have some effect in lessening the spread of the disease, particularly in normal years.

Since none of these three methods of directly attacking the disease meets our needs, we must consider the other alternative, viz: the fortification of the resistance of the pig. For this purpose we have available anti-hog cholera serum, which may be used either for wholesale, nation-wide immunization or only locally in infected or dangerously exposed herds. How can this serum be most effectively employed to prevent losses of hogs from cholera?

Let us consider first compulsory universal immunization. There is little doubt in my mind that such a system would effectively eliminate losses from cholera. But it would entail so many difficulties that in my opinion it is not at this time worthy of serious consideration. The only alternative to such a plan would be one based, primarily, on thorough but still quite voluntary cooperation of the farmers themselves. Such a plan was discussed and recommended by the late Dr. A. D. Melvin and the speaker, in 1917. Even after the lapse of ten years I believe that I cannot do better than again to urge the same plan, however, with certain modifications as I shall endeavor to explain.

Plan for the Control of Hog Cholera

Organization: A well-planned, smooth-working organization is essential to the success of any large undertaking. Such an organization is absolutely essential to effective control of losses from hog cholera. In a general way it seems that this organization should consist of three closely associated units:

(1) The federal government should enforce such reasonable regulations as may be necessary to lessen the chances of the spread of infection from public stockyards and cars or in any other way as a result of interstate commerce. In addition, the

federal government should work in cooperation with the various states, assisting them in the organization and education of farmers.

(2) Each state should maintain an organization, the prime object of which should be to enforce reasonable sanitary regulations such as restriction of the movement of sick hogs within the state and the proper disposal of carcasses of those which die of cholera. In addition, this state organization should have as one of its most important duties the organization of the farmers in communities for the control of hog cholera. It should endeavor to carry to every single farmer, knowledge of the disease and of the ways in which it may be controlled.

(3) The third unit should be the practicing veterinarians. In every community they should be the centers of farmers' organizations. These veterinarians should make it their special responsibility to maintain the health of the live stock in their own communities. It seems reasonable that they should maintain, as part of their regular equipment, ample supplies of anti-hog cholera serum and that they should let the farmers know how to reach them and how to obtain this serum when it is required. The interests of the veterinarian are best served by the prosperity of the farmer, and, if the practicing veterinarian will assume a practical responsibility for the prevention and elimination of disease in his territory, will form close contacts with the farmers and will let them understand that his purpose is primarily to help, I believe a long step forward in the control of hog cholera will have been made.

It will be seen that under this plan the state and federal governments would prepare the ground, so to speak, and lend support through the application of reasonable sanitary police measures, while the administration of the serum and the intimate contacts with farmers would be the functions of the practicing veterinarian. It may be said that this is the plan which is now being followed, and, in a general way, this is true. But I am inclined to think that the veterinarian is not getting close enough to the farmer. Too often we have heard of actual clashes of farmers' organizations on the one hand and veterinary associations on the other. I believe that the veterinarian himself can overcome this state of affairs by a better display of the real interest which he feels in the farmers' welfare and thus create a condition wherein the farmer, when he has trouble with his hogs, instead of writing to Washington, or to the state college, will telephone his good and

capable friend, the veterinarian. This close union of the farmer and the veterinarian seems to be at the very heart of any effort to control losses from hog cholera. With such a condition existing we could look hopefully to the future.

I shall not attempt to go deeply into methods which veterinarians should employ in combating hog cholera, though this offers a most alluring field for discussion. Is regular preventive inoculation to be practised or should dependence be placed upon sanitary measures with inoculation only after the disease has arrived? Should pigs be immunized before or after weaning? The answers to such questions must be worked out by each veterinarian so as best to meet the needs of each of his clients. There is much to be said in favor of regular immunization of all new pigs, but this is not always practicable. In some cases immunization of the suckling pigs is imperative, while in other cases it cannot be advised. There is continually some problem to be solved and, just as in human diseases, each case must be handled individually by a careful, well equipped professional man. It really makes very little difference how the pig is protected just so he is protected. There is only one general principle which it seems to me might with advantage be urged for universal adoption and that is the use of immune breeding stock. Not only would the producing reservoir be thus safeguarded but the pigs, owing to inheritance, would be more resistant to infection and less susceptible to so-called "vaccination breaks."

In concluding. I wish to repeat that the immunization of swine against cholera is a job for the practicing veterinarian. He should be supported by federal and state officers and will certainly be supported by the farmers of the country when they come to realize how greatly he can serve them.

Discussion

Dr. R. R. Birch: I have heard Dr. Dorset give a great many good addresses, but have never heard him give a better one than he gave today. I do not quite share his feelings regarding the futility of sanitary police measures, properly applied. Foot-and-mouth disease, well established, has been banished from this country several times, and rinderpest, thoroughly established, has been eliminated from South Africa and several European countries. We cannot compare these diseases too closely with hog cholera, but there is at least this difference. The campaigns against rinderpest and foot-and-mouth disease have been thoroughly in earnest; those against hog cholera have always accepted, as a matter of course, the probability of failure.

Dr. Dorset has outlined an admirable plan for the suppression of hog cholera. The chief advantage of the plan is its flexibility. The practicing veterinarian is on the spot to diagnose the case, to advise the man whether or when to vaccinate, which method to use, and regarding the influence of other diseases. As was pointed out, no two cases are exactly alike and no blanket measure will operate successfully.

If the plan Dr. Dorset proposes should go into effect, I hope special effort can be made to bring pressure to bear on three well-known channels through which hog cholera spreads from locality to locality. These are the shipping of infected hogs, the feeding of garbage which contains pork trimmings from slaughter-houses and even from packing-houses in which there is federal inspection, and the abuse of simultaneous treatment. If continued and effective pressure can be brought to bear on these channels the actual need for simultaneous treatment will be greatly reduced.

Please do not misunderstand the meaning of these remarks. If I were a farmer living in certain localities in Iowa, Kansas or Illinois, I would not think of raising hogs without maintaining the herd immune. Nevertheless, I do not think it is necessary that this condition shall always exist, provided greater care is taken to prevent the locality to locality spread of hog cholera regarding which I have just spoken.

DR. H. E. ASH: I would like to ask Dr. Dorset how long a pig from an immune mother will remain immune after birth, or how soon he might contract cholera.

DR. DORSET: I do not think that question can be answered definitely. It seems that either owing to difference in local conditions, or difference in the pig, or difference in the degree of immunity he inherits, the immunity he gets from his mother varies in duration. In some cases you will find pigs from immune mothers that are immune several months after they have been farrowed. In other cases, pigs apparently from immune mothers will be found susceptible in two or three weeks. It seems to vary. It is perhaps something we do not understand thoroughly. At the same time, anybody who has had experience with such matters will agree that, as a general proposition, pigs from immune mothers are more resistant than those from non-immune mothers, and certainly no one engaging in hog cholera experimental work to produce good virus would select pigs from an immune mother for such a purpose. The pigs from the immune sow, in the majority of cases, are immune some weeks after birth and after that the immunity fades away.

DR. ASH: It has been my experience in Ohio that as long as the pigs are on the mother we seem to have very little trouble. In 1926, of course, we had a big outbreak of cholera. This year our pigs developed hog cholera anywhere from two weeks on up and now the question in my mind is whether it is due to the virulence of the disease last year or whether the serum or virus we used on some of these sows possibly was not as good. The sows treated do not seem to develop hog cholera, but we are having a lot of trouble in small pigs this year. It is very acute. Now, how young can a pig be successfully treated and rendered immune?

DR. DORSET: I referred to this subject yesterday, discussing a paper on the immunization of young pigs, and I stated then that we immunized something like 5000 young pigs and we tested the immunity of about 2000 of those. They varied in age from ten weeks to one day. We waited, as a rule, on a number of farms, until all farrowing was completed, and then injected them all simultaneously. We found no difference in the immunity of those treated at ten weeks or those treated at one day. In other words, we found substantially that the very young seemed to acquire immunity quite as firm and as lasting as the older pigs. We found nothing to indicate that immaturity alone is a bar to the acquiring of immunity.

SIR ARNOLD THEILER IN AUSTRALIA

Sir Arnold Theiler left Basle, Switzerland, the early part of March, for Australia, at the invitation of the Commonwealth Council for Scientific Industrial Research. He expects to remain in Australia for about six months and will return to England via America before the end of the year.

THE CLINICAL DIAGNOSIS OF GENITAL DISEASE OF THE COW*

By W. W. WILLIAMS, *Springfield, Mass.*

Inasmuch as reproductive failure on the part of the cow is due to a wide variety of factors, it is highly important that the practicing veterinarian shall be adequately versed in the physiology and pathology of the reproductive system and otherwise equipped that he may make a reliable diagnosis. Unfortunately many veterinarians, even some termed specialists, have entered into practice dealing with genital diseases with little or no training in physiology, pathology or the technical training required for making a thorough clinical examination. Many have depended merely upon empiric treatments to obtain results. This has often led to gross clinical misinterpretations and misdirected treatment. With one man there may be the tendency to have all sterility due to a "closed cervix" and each sterile cow is subjected to a more or less crude cervical dilation; and in other cases, it is to a mania for ovarian troubles and all ovaries are "gently massaged" as the only treatment, although a careful perusal of the history and an intelligent examination of the ovaries would show clearly that the ovarian health is beyond suspicion. In other cases, we find aborting cows which are negative to the agglutination test for *Br. abortus*, considered healthy, although either a physical examination of the cow or a glance at the breeding record would quickly dispel any such idea. Instances where neither the case history nor a physical examination play a role in the matter of diagnosis are quite commonplace.

Diagnosis requires accurate and detailed information concerning the individuals involved and, in many instances, about the herd as a whole. It is important from the clinical standpoint that such information shall be concise, arranged in an orderly manner and to the point, eliminating useless and irrelevant matter. The average herdsman may require fifteen minutes for a discourse on the breeding history of an individual, whereas, when concise and pointed questions are asked and recorded on a chart, the essential information may usually be had in a few moments. By using a printed form on which the detailed records

*Presented at the sixty-fourth annual meeting of the American Veterinary Medical Association, Philadelphia, Pa., September 13-16, 1927.

of findings are made from time to time, one is better able to get a correct clinical picture, for memory is very unreliable when dealing with a large number of individuals. The record should include various relevant items in the matter of history, together

Owner..... Herd..... Case.....

Address..... DATE..... 192.....

COW'S NAME OR NUMBER..... Age.....

INDICATE REASON EXAMINED AND COW'S CONDITION BY AN "X" PLACED IN SQUARE.

1. ☐ Sterile. How long?..... Times served?
2. ☐ Retained afterbirth.....
3. ☐ Discard of pus..... How long?.....
4. ☐ Previous difficulty in breeding.....
5. ☐ Difficult labor.....
6. ☐ Gave birth to sick calf.....
7. ☐ Examine for pregnancy.....
8. ☐ Determine sexual health.....

CALVED..... 192..... No. of full term calvings.....

ABORTED..... 192..... No. of abortions.....

Interval between heats..... days. Character of menstruation.....

Duration of heats..... days. Character of heats.....

HEAT AND SERVICE DATES SINCE LAST CALVING. Place date in parenthesis if not served, i.e., (17)

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Year
192													192

REMARKS:.....

General condition..... Milk production..... lbs.....

DIAGNOSIS..... Herd..... Case.....

R. Ligament..... L. Lig.....

Vulva.....

Vagina.....

Cervix.....

Uterus.....

R. Ovary..... R. Tube.....

L. Ovary..... L. Tube.....

Treatment Applied.....

RECOMMENDED:.....

Heat due in..... days. Prognosis.....

W. W. WILLIAMS, D. V. M.
58 Bronson Terrace,
Springfield, Mass.

FORM 1. Examination form, showing reproductive history and condition of genital organs.

with detailed pathologic findings, all arranged in a manner that items of interest may be readily filled out and seen at a glance.

The general breeding conditions of the herd as a whole might well be considered first. One should know the make-up of the

herd, general conditions of individuals, whether assembled from miscellaneous sources or grown on premises, its previous breeding condition, the ratio of calf-production, the incidence of retained placentae, uterine discharges, sterility, etc. These are

Herd_____ Case_____

COW_____ Reg. No._____ Herd No._____

Born_____ 19___, Died_____ 19___, Cause of Death_____

Date entering herd_____ 19___, Sold for Breeding_____ 19___.

Name of dam _____

OWNER _____

Address _____

CONCEPTION DATES	CHECK IF ABORTION	CALVED OR ABORTED	BULL USED	DAYS PREG.	RTN PLAC.	DISCH OF PUS	REMARKS: HEALTH OF CALF, CONDITION OF COW, ETC.
19		19					
19		19					
19		19					
19		19					
19		19					

HEAT AND SERVICE DATES. Indicate menstruation by date in parenthesis, ie. (18).

19	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
Bred												
Bull												
Heats not bred												
Lbs. milk												

19	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
Bred												
Bull												
Heats not bred												
Lbs. milk												

19	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
Bred												
Bull												
Heats not bred												
Lbs. milk												

19	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
Bred												
Bull												
Heats not bred												
Lbs. milk												

FORM II. Breeding Record Form. Provides an orderly and systematic method of recording significant facts concerning the reproductive health of the cow over a period of years.

matters which often furnish definite clues as to the condition of a particular individual. We should consider methods established to prevent the spread of infection from one individual to another. Are healthy bulls used? Are maternity stalls adequate and

properly used? Are discharging cows given prompt and efficient attention? Bear in mind that genital discharges constitute the principal source of supply for pathogenic bacteria and, with

RECORD OF EXAMINATIONS.

NAME OF COW. _____

Diag. _____ Date _____ 19____.

Treatment _____

RECOMMENDATIONS: _____

Diag. _____ Date _____ 19____.

Treatment _____

RECOMMENDATIONS: _____

Diag. _____ Date _____ 19____.

Treatment _____

RECOMMENDATIONS: _____

Diag. _____ Date _____ 19____.

Treatment _____

RECOMMENDATIONS: _____

Diag. _____ Date _____ 19____.

Treatment _____

RECOMMENDATIONS: _____

Diag. _____ Date _____ 19____.

Treatment _____

RECOMMENDATIONS: _____

FORM III. For use when routine examinations of a herd is being made and only brief records are required.

this source eliminated, the most important step is taken in the matter of eliminating genital disease and its sequelae—abortion and sterility.

General Resistance

Another factor of very great importance is that of resistance of the individual or herd to infections in general. Resistance is a variable factor, and in many instances, accounts for the variability in reproductive ability of different herds or different individuals of the same herd, other factors being constant. Animals having insufficient exercise over long periods of time may generally be considered to be less resistant to infections in general and may succumb to infections which, under a higher state of resistance, would be harmless. Bacteriological investigations show that the organs of the non-pregnant cow ordinarily contain numerous bacteria and these same types of bacteria apparently assume a pathologic role under unfavorable influences. *Improper feeding* has a like effect upon resistance and one should consider the character and quality of foods used, whether a complete or a one-sided ration is being fed, also whether the feeder uses intelligence and discretion in his feeding the individual animals.

The veterinarian devoting his time to the preservation of the genital health of cattle is continually called upon to make many examinations for genital health before pathological conditions have asserted themselves by recognizable abortion or sterility. The mechanism of the production of the pathologic conditions should be understood whenever possible.

Recognition of Disease

An accurate history and a detailed physical examination furnish individually or collectively the most reliable means of diagnosis. It is the basis upon which the successful practicing veterinarian must rely for information to guide the prevention and therapeutics of genital disease. It furnishes information as to the severity of disease, the organs affected, the virulence of the infection, the resistance of the individuals and the type of pathology.

For the present purpose it seems best to outline the general indications of certain of the more commonly encountered pathologic conditions of the genital organs.

A general perspective of the cow's reproductive efficiency is obtained by a consideration of her age, the number of live healthy calves produced, the duration of gestations and the number of services required for impregnations. To determine the relation which poor function bears to tissue changes, a

résumé of various of the more commonly observed pathologies is necessary.

Vaginal Examination

A very small portion of reproductive difficulties are due to primary disease of the vagina, but vaginal examinations frequently furnish invaluable clues to significant disease located elsewhere. Thus, in examining the vagina, we should record the presence and character of any discharge. On entering the vagina, note should be made of the presence of Bartholin cysts and, further forward in the vaginal floor, enlarged Gartner's ducts, either cystic or abscessed, are occasionally observed.

A very insidious form of sterility which, however, responds readily to surgical treatment, results from abscessation of Gartner's ducts. Even when making pregnancy examinations, vaginal inspection is important. It allows the detection of the cervical seal of pregnancy which is a very important item as regards the safety of pregnancy, and frequently allows the recognition of an abortion in its early stages which otherwise would escape detection. A great many cows abort prior to the third month and, being unobserved, do not receive attention. Frequently membranes of four to six weeks' development are detected upon vaginal examination and I am fairly convinced, from numerous observations in this respect, that abortion during the first three months of gestation would be quite as great as during any other period, provided the fetal movements did not, during the latter months, bring about mechanical injury of the placental attachments and thus furnish a more fertile field for the growth of bacteria. Judging from the high death-rate of the ova of multipara prior to uterine implantation, it seems reasonable to assume that there is also a high mortality of the unimplanted but fertilized bovine ova.

Disease of the Cervix

In herds where abortion and sterility are a common feature, cervical inflammations constitute usually the most prevalent lesion. Cervicitis often interferes with the formation of the mucous seal of pregnancy, thus inducing abortion and, when present in the non-pregnant animal, is a frequent cause of sterility.

The history gives little or no indication of the disease. In some cases, there may have been a continual purulent or muco-

purulent vaginal discharge, but the character of discharge is not sufficiently typical to identify its origin in any way.

In cases of difficult labor, due to an oversized fetus or insufficient relaxation of the birth canal, cervical lacerations and inflammations may be suspected, but the best means of diagnosis is a careful physical examination.

Examination of the cervix is facilitated by retraction with the aid of a pair of Albrechtsen forceps. One observes whether the folds are of a normal pinkish color, the character of any discharge, the presence of any chronic inflammation, lacerations, nabothian cysts, etc. But a vaginal examination usually brings into view only the lips and first cervical fold. The enlargement of the first fold, even in the absence of an inflammation, suggests the possibility of disease further forward, thus requiring in many cases that the examination shall include the anterior folds. If the cervix is not too small, a slight dilation of the lips and posterior fold may permit observation further forward. Otherwise, an attempt should be made to detect any induration by rectal palpation. Lacerations of the cervix leading to cicatrices, hypertrophy and ectropion often occur. Provided, however, these enlargements are non-inflammatory, they may exist without clinical significance. Yet their mere presence does render the cervix more prone to future disease.

It has been stated that examination of the cervix can best be made with the aid of a vaginal speculum. To this, I cannot wholly agree. If the speculum alone is used, the examination may be too superficial. Only the lips or perhaps the first fold is observed. It is claimed, and perhaps justly, that the application of forceps induces a congestion, giving a false impression of cervicitis. If forceps are applied to the vaginal side of the cervical lip, never applied to a cervical fold, and observation made at once, a confusing congestion of the cervical mucosa may be avoided. A vaginal speculum is often a convenient instrument to have, but as a whole, it adds little or nothing to the accuracy of diagnosis.

"Closure of the cervix" is a condition diagnosed much, but little observed. Closure of the cervix signifies a stenosis which prevents the passage of fluid through the cervix and in these cases menstrual débris collects within the uterus. It is quite safe to say that veterinarians who have treated closure of the cervix the most, have observed the condition the least. Functionally, however, the cervix is frequently, to all intents and

purposes, closed by inflammation, preventing the migration of sperms or otherwise interfering with its physiologic duties. A swelling and induration of the anterior fold often makes entry into the uterus difficult, especially with heifers.

Endometritis

When the endometrium alone is involved, tissue changes are usually insufficient to allow clinical recognition by physical examination. When a uterine discharge exists, we may at once presume an endometritis. Otherwise, it is necessary to base our diagnosis upon such history as the placenta health, the health of the fetus at the time of birth, the presence of an abnormal uterine discharge at any time, but, most important of all, is the condition of the placenta. This organ furnishes the best evidence of health or disease of the endometrium and should be examined whenever possible. Unfortunately, the placenta from sterile animals has usually not been obtained and it is therefore often impossible to determine its health. The presence of an endometritis may be presumed when endometrial infections are known to be very prevalent in the herd in question, even when physically and functionally the genital organs appeal normal. In many such cases, prompt conception has followed uterine disinfection. This has been outstanding upon several occasions where groups of apparently normal heifers in a given herd have been sterile.

It is possible to make a direct visual examination of the endometrium by the combined use of an endoscope and uterine insufflation, but I am not aware that this mode of examination has to date received any attention.

Williams, Udall, Frost and others[1] have pointed out that with endometrial injury the development of the placenta is impaired and, by applying this principle in a practical manner, one is frequently enabled to recognize the cardinal symptoms of endometritis by the subnormal development of the non-gravid cornu during the early stages of pregnancy. Impending abortions may therefore be recognized in some cases very early in gestation.

Metritis, Pyometritis, Perimetritis

Metritis and pyometritis usually present no great obstacles to diagnosis. Metritis following parturition commonly inhibits involution. In acute types, a rise in temperature and various systemic changes may be noted. A rectal examination of the

uterus usually distinguishes the condition. Pyometritis is usually recognized on rectal examination, although when only a few flakes of pus are present, uterine douching may be the only method of detection. Whenever pyometritis is suspected, it is well to douche the uterus, for diagnostic purposes, if no other. The presence of vaginal pus indicates pyometritis, but care needs be taken that the pus formation is not entirely due to a vaginal or cervical disease for, in such instances, it is not policy to enter the uterine cavity and thus provoke an upward extension of the infection. Perimetritis is usually the result of a mechanical injury to the uterine wall and may be detected by the peritoneal adhesions which are provoked.

Uterine Tumors

Uterine tumors occur occasionally and may be recognized readily upon rectal examination. The most frequently encountered are due to hypertrophied maternal cotyledons which have not undergone complete involution, cystic endometrial mucous glands and tuberculous nodules. Occasionally, uterine abscesses are encountered. Their particular characteristics usually distinguish them from one another.

Salpingitis

Salpingitis is apparently usually the result of invasion by bacteria from the uterine cavity. Its diagnosis is of importance owing to the high incidence of sterility and many times abortion, for the tube furnishes a remarkably permanent habitat for invading bacteria. Tubal disease is common following a severe uterine infection, especially following parturition, but the virgin heifer by no means escapes. Clinically the most prevalent form is due to a non-purulent infection, but types of infection vary in different herds. In some a pyogenic form prevails.

An intimate knowledge of the genital health of the herd in general may produce valuable information for the diagnosis of tubal disease, for if the type of infection present in a given herd tends to have a particular affinity for the tubes, we are at once warned of their possible invasion. A pyogenic salpingitis is invariably fatal to procreation, while many cases exhibiting only a mild exudative endosalpingitis may be expected to recover under proper therapeutics, especially when not featured by bilateral tubal cysts or perisalpingitis.

Recognition of tubal disease is dependent almost wholly upon a careful physical examination. For this, a first-hand knowledge

of the anatomy of the adnexa must be had and the diagnostician must have sufficient experience in the matter of rectal palpation to interpret what he feels. The normal oviduct of the cow is readily palpable for its entire length. This is most readily accomplished by following the tube from the isthmus distalwards, the thumb on one side of the mesosalpinx and the first and second fingers on the other. In this manner, exceedingly small tubal cysts may be detected; and, as the thumb passes lateralwards over the anterior face of the ovary, into the ovarian pocket, adhesions of the tube or of its fimbriated extremity may be detected. Better still, the distal end of the tube and its pavilion may be palpated by passing the index finger into the ovarian pocket, in which position the pavilion and ostium abdominalis may be brought readily between the thumb and index finger. In so doing, tubal cysts as small as $\frac{1}{16}$ to $\frac{1}{8}$ of an inch in diameter may be readily recognized. Strands of connective tissue, extending between the pavilion, mesosalpinx and ovary, are easily distinguished. Occasionally a parasalpingitis, para-ovaritis and parametritis result in general adhesions of the tubes to the adjacent peritoneum, and then it may be impossible to trace the course of, or even palpate, the tube at all. A frequent indication of tubal disease is the presence of a cystic corpus luteum, a pathologic condition due to ovarian infection and which is apparently, in many cases, secondary to an infection of the tubes.

It happens occasionally that the fallopian tube is completely blocked by a chronic productive inflammation, and although it exhibits no abnormalities, either to touch or to the naked eye, it is entirely impossible for ova or spermatozoa to pass through. Other factors being normal, it is therefore important that tubal patency be determined. For this, I have been using a uterine insufflation apparatus originally described by Dr. G. L. Moench, of the New York Post Graduate Medical School. In the cow, gas will flow through the normal tube under a pressure as low as 25 to 35 mm. at a rate of about one milliliter per second. A description of the apparatus for uterine insufflation appeared in the *North American Veterinarian*.[2,3]

Ovarian Disease

Ovarian sterility may be divided roughly into two main groups, that resulting from endocrine dysfunction, and due to the improper development of the follicle. Owing to the close relation existing between these and the constitutional health of

the cow, the clinical picture of ovarian disease is often confused. An infection of the ovaries is often at the root of the trouble in either case.

Three anatomical components of the ovary must be considered, the germinal portion, the interstitial ovary, and the corpus luteum. Infection as a rule causes sterility by interfering with the development or liberation of the ovum. The observed effect is usually an increased heat interval or the absence of heat. This is accomplished usually either by the development of an oöphoritis or peri-oöphoritis, wherein an induration of the ovary or a thickening of its peritoneal coat prevents the rupture of the follicle; or the absence of heat may result from an infection of the crater of the ruptured follicle and the subsequent development of a cystic corpus luteum which does not readily undergo lysis.

In the diagnosis of an ovarian infection, we must first consider the probability of an ascending infection. Primary ovaritis is apparently rare. Oöphoritis or involvement of the matrix of the ovary, when sufficiently developed, may produce an induration of the ovary not dissimilar to the ovarian condition commonly observed amongst very old cows; and this induration inhibits the formation of the follicle. Upon palpation, the ovary presents a firm, though unevenly pitted texture. Frequently, by a little pressure, numerous little atretic follicles may be ruptured, a condition which does not occur with the normal cow.

Peri-oöphoritis may usually be recognized by the presence of adhesions of the ovary to the adjacent peritoneal surfaces, usually the tubal fimbriae and mesosalpinx being involved. Infection of the corpus luteum is usually distinguished by its cystic degeneration. A cyst forms in the middle of the lutein tissue, causing an enlargement of this body. In some cases, the fluid becomes so great that it is mistaken for a cystic follicle, but by careful palpation, one may be able to distinguish a little lutein tissue. Aside from this, the fact that a cystic follicle tends to produce nymphomaniac symptoms, while a diseased corpus luteum tends to lengthen heat intervals, usually distinguishes the two. Cystic degeneration of the corpus luteum also should not be confused with the presence of a lutein blood-clot which is frequently observed for a few days following estrum.

Functional Disease of the Corpus Luteum

There is no other portion of the genital system of the cow which has been so subject to the devices of the empiric as the corpus luteum. It is because so many have entered the clinical work on genital diseases without sufficient preparation in the physiology or pathology of the reproductive organs. The physiologic role of the corpus luteum is often insufficiently understood and the owner of cattle is led to believe that its mere presence is a pathologic condition. In the first place, we can usually dispense with the probability of luteal disease in the presence of regular, normal heats. Retention of the corpus luteum is a common incident with the absence of estrum, yet the frequent examination of cows with a reported failure to come in heat, leads me to the belief the failure of the care-taker to observe heats accounts for the major portion of the cases whose failure to come in heat is supposed to be due to a retained corpus luteum. The distinguishing anatomical condition of a retained corpus luteum is usually an abnormal protruding of the fully developed lutein body from the surface of the ovary, or, in other cases, its embedment in the ovary, the latter occurring more particularly with pyometritis, mummification and early fetal death. In either case, it tends to be more firmly attached to or embedded in the ovary than pertains to the normal corpus luteum. In my experience, retention of the corpus luteum is rarely, if ever, of primary origin. It occurs under such circumstances as pyometritis, early fetal death, mummification and various constitutional or general disease conditions in which the genital functions are influenced. It is a common feature in the presence of peri-oöphoritis or cystic degeneration of the corpus luteum. It is always important that any primary condition as a cause for retention of the corpus luteum or absence of estrum shall be recognized as a guide to therapeutics.

The Corpus Luteum and Estrual Cycle

The view is expressed by some writers that the corpus luteum is the regulating mechanism of the estrual cycle. A more accurate view of the matter seems to be that the duration of the live ovum is the major regulating device under natural conditions, but under disease conditions, where lutein lysis is inhibited, estrum may also be inhibitied. It is also apparent that the corpus luteum is not necessarily inhibitory to heat. It does perform certain important functions in the estrual cycle and

apparently inhibits maturation of further ova. However, upon the death of the ova or upon the termination of gestation, lysis soon brings to an end the inhibitory influence.

It is interesting to note that the injection of lutein solution usually has no apparent effect upon the induction of estrum. Although having used corpora lutea solution with a great number of cows, with injections spread out usually over a period of five to eight weeks, I have had no indication that the estrual cycle has been influenced. The dose used has been one or two ampules every day or two. Perhaps larger doses might have produced a different result.

Disease of the Follicle

Disease of the follicle bespeaks disease of the ovum and, at least in the clinically recognizable cases, is usually indicated by disturbances in the estrual cycle. Atresia of the follicle principally occurs in heifers, although at times in old cows which have passed their normal breeding age. In the heifer, heats often occur at too frequent intervals and, upon palpation, the ovaries are usually found to contain numerous small follicles. These follicles usually develop sufficiently to induce estrum, then fail to rupture and, within a week or two, the same process may be repeated. Corpora lutea are usually exceedingly small or fail to develop. In old cows, the same process is often noticed, but here there is frequently an insufficient development to induce estrum.

In some cases which have been examined at frequent intervals, I have been unable to discern either follicles or corpora lutea, thus indicating a non-development of follicles or ova. Undoubtedly the microscope would class such cases as atretic follicles, but some of them do not develop to sufficient size to be palpated. Follicular non-development is occasionally met with in poorly nourished and poorly developed heifers. In one instance where most of the cows in a herd of forty failed to show heats, I could recognize neither follicles nor corporea lutea in most of the animals. The herd was in an extremely poor general physical condition from improper and insufficient feeding.

Cystic ovaries or nymphomania is a term commonly used to indicate a syndrome, or aggregation of symptoms, commonly observed in the cow, in which the failure of the follicle to rupture, together with an excessive collection of follicular fluid, is a prevailing feature. The symptoms of this disease such as the

relaxation of the sacro-sciatic ligaments, swelling of vulva, atony of vagina, cervix and uterus, ovarian cysts, absence of a corpus luteum, an abnormal sexual desire, continuous heat, development of male-like characteristics, etc., distinguish it from all other genital conditions, so that little difficulty is experienced in its diagnosis.

For a differential diagnosis in early cases, one needs be sufficiently versed with physiology, pathology and the physical examination of the genital organs to distinguish such anatomical structures as a cyst, follicle, mature ovisac and corpus luteum, and the normal cyclic changes of the ovary must be borne in mind. There is in many quarters an evident lack of effort in distinguishing ovarian conditions, with the result that many cows with perfectly normal functioning ovaries are considered as cystic.

Sterility with Negative History and Clinical Findings

When examining cattle for sterility, it frequently happens that perfectly sterile cows present a normal history and no physical indications of disease. The practitioner who is called upon to examine such animals feels duty bound to give them some sort of treatment in order that they may become fertile, but the fact that treatment is in the blind renders this group of cases the least satisfactory to treat and produces a low ratio of recovery.

As a cause for these negative cases, we may consider principally such factors as a mild endometritis, an endosalpingitis, or a tubal stenosis. It is well to try one line of treatment at a time, for it often happens in a given herd that sterility of different individuals trends toward the same cause. It is important, therefore, that this cause be determined as an aid to the treatment of other individuals. A plan which I have been following in such cases is first a uterine disinfection, rather as a matter of routine. A careful check is made upon the general condition of the cow, her food, exercise, etc. If faulty, the necessary corrections are made. Then, if she still fails to conceive, I have used (and in many cases with good results) corpora lutea solution intramuscularly. This substance induces a premenstrual endometrial engorgement and is supposed to aid in the implantation of the ovum. In other cases, uterine insufflation with carbon dioxid gas has proven that the oviducts are closed and that the cow is in fact sterile. In a few instances, insufflation has had

an apparently valuable therapeutic effect, for the cows so treated have conceived at the next service following the forcing of the gas through the tubes.

Endocrine Pathology

Owing to general reflection of systemic disorders upon the functioning of the generative organs and reproduction, endocrinology has attracted considerable attention from the gynecologist during the past few years, especially in relation to uterine and ovarian dysfunctions. It is almost needless to say that the action, uses and indications of most of the endocrine products are almost unknown; but this has not prevented unscrupulous proprietary houses from forming various endocrine mixtures for all manner of genital disorders, nor has it prevented many practicing veterinarians with no training in endocrinology from using their products.

The symptomatology of endocrine disorders affecting the genital system of the cow is very poorly defined. Current veterinary literature has produced little of scientific merit along these lines, most of it arising from purely commercial sources. The principal advice which I would venture to those wishing to delve into endocrinology is to procure some standard works on the subject, or perhaps to subscribe to the *Journal of Endocrinology* which, aside from its numerous original articles, makes a critical review of the chief literature on the subject.

Unless the veterinarian is willing to make a real study of endocrinology, he is working a mere commercial hoax upon his clients in the use of the various products. If he is studying the subject, he will use the various products individually according to individual indications and use them long enough to observe their physiologic action. Then, if more than one endocrine product seems indicated, he will compound according to the individual requirements. First, however, it is necessary to obtain a working knowledge of the physiological actions of the various glands and that cannot be had from the bottle-labels and advertising literature of patent-medicine houses.

Some of the actions of and indications for a few of the endocrine products, such as thyroid extract, adrenalin and pituitrin, are well understood, but here one should distinguish between the truly endocrine behavior of the product and its pharmacodynamic result. Most veterinarians are familiar with the pharmacodynamic effects of adrenalin chlorid and pituitrin, but the

constitutional effects which are obtained by their use, over a long period of time, provide an entirely different problem.

REFERENCES

[1]Williams, W. L., Udall, D. H., Frost, J. N., Goldberg, S. A., Cushing, E. R., Fincher, M. G., & Mabey, M. H.: Corn. Vet., xiv (1924), 4.
[2]Moench, G. L.: No. Amer. Vet., vi (1925), 10, p. 25.
[3]Williams, W. W.: No. Amer. Vet., vi (1925), 11, p. 17.

DISCUSSION

DR. D. H. UDALL: My only excuse for discussing this paper is that my name was written on the program.

Dr. Williams has mentioned a system of keeping records. Various forms have been prepared, but the one that I fine most convenient is a ruled sheet, 6 x 9½ inches, with vertical red rulings about one-half inch apart. These sheets are punched to fit a note book. In the left margin, the year is written; in the left column, the blood examinations; in the second column, the heat dates; in the third column, the breeding dates and bull; in the fifth column, the date when the cow was diagnosed as pregnant; in the sixth column, the termination, leaving a margin of about two inches for remarks. Details of treatment may be placed on the reverse side. One sheet will contain the breeding information for the life of the cow, and it is available at a glance.

The speculum is useful in the diagnosis of pregnancy. Sometimes a cow is said to be in heat, when the speculum reveals the absence of mucus in the vagina and the absence of estrum. Professor Benesch brought from Vienna a speculum that we have found very practical because of the ease of application and the rapidity of adjustment. The view of the cervix as seen through the speculum is quite different from that of the same cervix after it has been grasped in the forceps.

In douching the uterus for diagnostic purposes, like Dr. Williams, I follow this method as a routine, watching the hairs at the ventral border of the vagina for flecks of pus that come from the uterus and indicate endometritis. Douching the uterus is sometimes claimed to cause salpingitis, but I have not seen evidence of such an effect. It is probable that such claims are based on theory rather than observation.

Failure of heifers to breed is a topic upon which Dr. Williams has given some interesting opinions. Any explanation of this common defect is useful, for too often we are unable to find a cause. With the growth of knowledge on the subject, we shall probably find increasing evidence to support the claim of W. L. Williams, that successful breeding depends on healthy genitals and a healthy growth. In herds where the histories are known, one finds abundant evidence that disease, or poor nutrition, in calves is a forerunner of sterility.

DR. H. E. BEMIS: I want to compliment Dr. Williams upon the thoroughness of his methods and the introduction of new methods in diagnosing certain conditions affecting the uterus and tubes. I think that such work is distinctly in the right direction. Although certain methods may not come to be of general usefulness, they will help out in those cases where the diagnosis is not clear and where the value of the animal warrants the use of such methods.

A knowledge of the physiology of the whole genital tract, as it is tied up with the function of the entire animal body, is, as he said, most important. A study of the herd history; a study of the animal itself; a study of the blood lines represented in the individual and a knowledge of the performance of the ancestors may throw light upon what may be expected of the offspring. Early cessation of breeding may be a family characteristic. The conformation of the animals in question, especially if it tends towards masculinity, is another point to be observed.

I agree with Dr. Udall concerning the use of the vaginal speculum. I think there is much less injury done when it is used; the tissues are in their normal position and show their normal colors. The use of a diagnostic light will show those structures and make unnecessary any manual entrance into the vagina in a great many cases.

I believe Dr. Williams did not mention the accumulation of a lymph-like fluid in the uterus, which we find to be quite common and which often compli-

cates the diagnosis of pregnancy, the uterine wall being extremely thin, somewhat similar to the feel of the wall in case of pregnancy. In that case there is also a retained corpus luteum. The treatment of this condition is the same as in pyometra, as far as evacuation of the uterus is concerned, viz., removal of the corpus luteum. Often no other treatment is necessary.

There were some statements concerning the function of the ovary, in connection with ovulation, and the function of the corpus luteum, which we might discuss but which I think we will not discuss. I believe it was stated that the animal might not come in heat even though she had ovulated. Our anatomists, with whom we have been working, tell us that ovulation does not take place until 30 to 60 hours following the first appearance of heat.

Another question in my mind is that if the corpus luteum does not withhold heat, why does heat not occur in the case of retained corpus luteum?

DR. W. L. WILLIAMS: I shall make but one or two remarks. In the first place I think that the writer has brought very clearly before us the necessity for extensive study in order to make safe diagnosis with reference to genital disease. It has been entirely too much a rule-of-thumb practice and too few veterinarians are willing to take up the study in earnest and place that amount of work upon it which is necessary in order to make reasonably accurate diagnoses. After one has studied the subject for a great many years, at least, judging from my own experience, there still remains an abundance of room for error in diagnosis. Although a man never becomes so experienced that he does not occasionally make errors, certainly with experience and earnest study he lowers the frequency of these and we can reach a point in the diagnosis of genital diseases where his diagnosis is as reliable as it is in other fields of work.

Regarding the douching of the uterus as an aid to diagnosis and the danger that it may induce inflammation of the oviducts or force bacteria into them, I think that there is probably some danger when one distends the uterus very greatly, as was formerly done in some cases. I do not see that the ordinary use of the antiseptic douche for diagnostic purposes, where there is no material pressure placed upon it, and a reflow tube is used, is dangerous. At any rate, the usefulness of this in diagnosis far outweighs, under proper precautions any danger which may accompany it.

BUREAU TRANSFERS

Dr. H. S. Perdue (Iowa '15), from South St. Paul, Minn., to Cedar Rapids, Iowa, on meat inspection.

Dr. Edward Lapple (Cin. '11), from Fort Dodge, Iowa, to Indianapolis, Ind., on virus-serum control.

Dr. Herbert C. Berger (Cin. '17), from Indianapolis, Ind., to Cincinnati, Ohio, on meat inspection.

Dr. Laurence V. Cummings (K. C. V. C. '15), from Omaha, Nebr., to Fort Dodge, Iowa, on virus-serum control.

Dr. John H. Lowe (Iowa '00), from Omaha, Nebr., to Helena, Mont., on field inspection.

Dr. Wilber C. Finn, from Sioux City, Iowa, to Omaha, Nebr., on virus-serum control.

Dr. Wm. L. Endsley (K. C. V. C. '10), from Wichita, Kans., to Fort Worth, Texas, on tick eradication.

Dr. Ralph V. Pilgrim (K. C. V. C. '15), from Little Rock, Ark., to Fort Worth, Texas, on tick eradication.

Dr. Guy L. O'Harra (K. C. V. C. '18), from Chicago, Ill., to Ottumwa, Iowa, on meat inspection.

Dr. Maurice S. Shahan (Colo. '24), from San Francisco, Calif., to Sacramento, Calif.

Dr. Harold J. Boyce (K. C. V. C. '17), from Topeka, Kans., to Kansas City, Kans., on virus-serum control.

Dr. Floyd S. Ratts (K. S. A. C. '22), from Kansas City, Kans., to Topeka, Kans., on virus-serum control.

SOME PROBLEMS FOR THE PRACTITIONER IN COMMERCIAL DAIRY HERDS*

By E. R. CUSHING, *New Brunswick, N. J.*

One of the greatest problems with which the practicing veterinarian is called to cope, in dairy herds, is that of mastitis and in large dairies it becomes a real economic factor as well as a health problem.

In a certified dairy of about 250 animals which is under our veterinary supervision, the problem of mastitis is a grave one and I know, from talking with veterinarians connected with other large herds, that their mastitis problem is very similar to ours. In certified herds under constant supervision, where the fore milk is drawn through fine strainers, we see these cases sooner than in herds producing a lower quality of milk. In many of the latter, a hard quarter or a few curds in the milk has no particular significance and if they do assume symptoms of sufficient importance to alarm the owner, he exhausts all of his efforts and those of his neighbors before consulting his veterinarian. By this time, the case has advanced to such a state that the prognosis is very poor, even under the care of the most skillful practitioner.

The chief types of infection producing mastitis in our locality are streptococci and staphylococci, with occasionally *B. coli*, *B. necrophorus* and others. I will not attempt to classify mastitis according to etiology, as I am not a bacteriologist, but I will say that it is my experience that streptococcic mastitis is more likely to recover without leaving indurations in the udder and often responds to treatment more readily than does a staphylococcic mastitis. A streptococcic mastitis of very long standing, however, is frequently followed by atrophy, to a greater or less degree, of the involved portions of the udder.

We see mastitis appearing under a wide variety of conditions, some of which have some special significance. For instance, we frequently see this picture: A cow, for no apparent reason, may suddenly show partial or complete anorexia, with one or two quarters of the udder hot, swollen and giving coagulated milk. With prompt treatment, she may make an apparent recovery in two to five days and take her place again in the milking string, only to have the same symptoms reappear in from a few days to

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a month. It has been our experience that the majority of these cases have traumatic gastritis and peritonitis and eventually show more severe symptoms, with death finally ensuing from this cause.

Again we see cows freshen with mastitis or develop symptoms of it soon after calving. If the patient has been kept in clean surroundings, minimizing the danger of outside infection gaining entrance through the teat-canal, we then expect one of two things —either she has a history of previous mastitis or else she has a severe metritis. The latter condition is very frequently seen and the accompanying mastitis is slow to respond to treatment until after the metritis is well taken care of.

Another condition under which mastitis appears with us, and one which gives us no little concern, is the case of mastitis which appears suddenly in the apparently otherwise healthy cow in the certified string. In spite of close supervision, some milkers will have more mastitis in their string of cows than will others. This I think can frequently be attributed to faulty care and particularly to failure to milk their cows dry. One large dairy concern has adopted the practice of stripping the cows after the milkers have finished. By stripping into a strainer-cup, they frequently detect mastitis cases not showing evidence in the fore milk.

In a certain certified herd, we have had more mastitis cases during the hottest weather than at any other time. Whether this is merely a coincidence or may be partially attributed to weather conditions, we are unable to state. One herdsman is quite convinced, from repeated observations, that the feeding of freshly-cut, green alfalfa during hot weather is responsible for a coincident increased amount of udder infection.

As a prophylactic measure in combating this trouble, in a few instances of streptococcic infection, we have used repeated injections of an autogenous living streptococcic vaccine made from organisms isolated from affected milk. We have given an initial dose of 0.5 cc of the living culture subcutaneously, followed at 48-hour intervals with 1 cc, 1.5 cc and 2 cc. The results have been promising in some instances but not sufficiently conclusive to warrant regarding this method of prophylaxis as always successful. Several large dairy concerns have done some work along this line and possibly veterinarians connected with these companies are in a position to give more definite views concerning its feasability.

In the general routine treatment of mastitis, our first procedure is to administer a purgative, stop the feeding of all high-protein grain mixtures and substitute bran or oil meal. If we can see the patient at the first signs of udder trouble, we generally administer 1 to 1½ ounces each of formalin and turpentine as suggested by Dr. Frost, and repeated in 12, 24 and 36 hours, if necessary. We feel that this latter medication is of most value in cases that we can see soon after their onset. We also make cold applications to the udder in the summer and hot applications in the winter, followed by massage with some penetrating liniment. We advise frequent milking of the affected quarter.

If the patient shows general symptoms such as high temperature and pulse rate, we usually give 80 to 100 cc of a 1-500 solution of acriflavine intravenously, repeated in 24 and 48 hours if necessary. This has given excellent results in our practice in controlling bacteriemia and septicemia coincidently with udder infections.

In the treatment of staphylococcic mastitis in herds where several cases have been encountered, we have used autogenous bacterins consisting of a relatively heavy suspension of dead organisms. As an initial dose, 5 cc has been injected subcutaneously, this dose being repeated in 24 and 48 hours. After subsequent intervals of 48 hours, the dose is increased respectively to 8, 10, 12 and at times 15 cc. This method of treatment has given the most satisfactory results in this type of infection.

In herds where streptococcic mastitis is prevalent, we have used autogenous vaccines, which were heavy suspensions of the living organisms, as a curative agent, employing 0.5 cc as an initial dose, injected subcutaneously. This dosage is repeated daily for four days, after which 1 cc is given over a similar period of time. At four-day intervals, the dosage is increased until 3 cc is given daily as a maximum dose.

The subcutaneous injection of suspensions of living, freshly isolated streptococci may appear theoretically to be fraught with great danger. However, it must be recognized that there is a wide variation in the virulence of different strains of streptococci. In our experience so far, this method has not produced any harmful results, but, on the contrary, the results have been noticeably beneficial.

Metritis and Allied Affections

Genital troubles such as metritis, retained placenta and allied affections, constitute another problem always present in assem-

bled commercial herds. Volumes have been written and years of study devoted to this subject and I will therefore touch on it only briefly.

Every practitioner has a particular method of handling this problem, which has worked out more or less satisfactorily in his hands. In handling cases of acute metritis, the veterinarian must exercise considerable judgment, and his procedure must be dependent upon the symptoms presented. Some men prefer daily douching of the affected uterus, but this has not proven entirely satisfactory in our hands. If the general condition of the animal leads us to believe that there is an accumulation of debris and exudate in the uterus, we generally syphon this off and douche with hot sterilac solution ½ to 1 per cent, providing this occurs within two or three days subsequent to calving. If a longer time has elapsed since calving, before the condition is noticed, frequently the uterine wall is weakened to such an extent that much manipulation is dangerous. In this condition, the exudate is syphoned off with the least possible disturbance to the uterus, using a soft rubber catheter.

After removing the accumulated exudate from the uterus, the patient is given subcutaneous injections of 3 to 4 cc of pituitary extract twice daily over a period of three or four days, or at least until rectal palpation shows that the uterus is reducing in size and increasing in tone. At this time the character of the exudate generally changes from a reddish or brownish color to a yellow purulent material. This generally indicates that the acute metritis is subsiding and the danger of general septicemia is materially lessened. After this stage, we douche the uterus more frequently, using ½ to 1 per cent hot sterilac solution. If the uterus is still large and flabby, pituitary extract is continued. In the subsequent handling of cases of metritis, it is our practice to douche the uterus during or immediately following the first estrum. If there still remains some evidence of purulent exudate at this time, the uterus is treated accordingly.

The treatment of acute metritis by the use of pituitary extract may seem to some as prohibitive on account of the expense involved. In the herds where we are using it, strict accounts are kept and we have found that the cattle are ready to go into full production in enough shorter time and in a much better condition, to more than warrant this expenditure. In this connection it must be borne in mind that there is a wide variance in the relative strength of pituitary extracts obtained from different

sources. This fact emphasizes the necessity of purchasing the product only from reliable concerns who carry on physiological standardization. Pituitary extract U. S. P. X is three times as strong as pituitary extract U. S. P. IX and consequently this fact must be borne in mind when making purchases. We use the stronger solution.

Digestive Disturbances

A third problem in large herds is that of digestive disturbances. This subject presents a different aspect in large commercial herds than it does in the ordinary farm herd. In the latter, digestive troubles are more or less seasonal, occurring with greater frequency as the length of time away from pasture increases. Primary digestive troubles are at a minimum when the animals are on pasture. In large herds where the cattle do not have long daily access to pasture but are dependent upon barn feeding, digestive disturbances are ever present to a more or less degree. Primary digestive disorders generally respond to the ordinary methods of handling but where the trouble is secondary to traumatic gastritis and peritonitis, medical treatment is not satisfactory. The question of traumatic gastritis, peritonitis and pericarditis is a serious one in assembled herds and as yet no entirely satisfactory method of handling it has been brought out. It is my personal opinion that many cases of apparent primary digestive disturbances are actually secondary to this condition. This opinion is based upon subsequent postmortem findings in cattle which have been sold for various reasons, for slaughter.

In purely commercial herds, especially in regions near the larger cities, it was long the custom to buy cows which were fresh or approaching termination of gestation and keep them until the end of that lactation period, at which time they were sold for slaughter. This practice is still pursued by some dairymen but most producers have been forced to adopt another procedure—that of breeding their better producers and maintaining them in the herd as long as they are profitable. Even with this policy, the ranks of the producing herd are depleted all too rapidly and the dairyman is confronted with the problem of replacements in order to maintain a constant milk supply.

A few years ago a man could easily purchase desirable cattle at a moderate price but today it is becoming difficult to purchase the right kind of cattle at any price. Thus the dairyman is confronted with the problem of raising a part, if not all, of his own

replacements. In many ways this will increase the efficiency of his herd in as much as he will raise the offspring of only the better producers. This procedure, of course, entails an additional investment, accompanied with many additional problems to be solved.

In the certified herd before mentioned, where cost accounts are carefully computed, they have been raising part of their replacements for several years. Their figures show that, exclusive of the labor entailed, their young stock represents an investment of about $110 to $115 a head, at the age of 24 months. The labor cost, of course, is variable, according to location, but even in the metropolitan area, with high labor charges, their two-year-old heifers do not cost much more than $175 apiece. Then, too, they have a better quality of cattle than that figure will purchase in the open market.

Calf culture in these herds places added responsibilities on the attending veterinarian. It has been our experience that the adoption of systematic prophylactic measures against calf diseases is much more satisfactory than the treatment of the diseases after they are present in the animal. After experimenting with many methods or systems of feeding young calves, we have adopted, in a general way, the method as brought out a few years ago by Dr. Udall, as the most satisfactory. In any method of caring for calves, it is important that the calf be born in a clean place—a clean box-stall or the pasture. Briefly his method is as follows: The calf is allowed to remain with the dam for a period of 8 to 12 hours, depending upon the activity of the youngster, in order that it may receive the colostrum which is regarded as very important. The calf is then removed from the dam to a separate clean stall or pen, weighed, and a muzzle placed over its mouth. All additional food is withheld for 15 to 24 hours, after which it receives one pint of milk and one pint of lime water, warmed to 100° F. and fed in a sterilized pail.

The next day the animal receives 5 per cent of its body weight, of milk, divided into three equal feedings and to each feeding is added one pint of lime water. Each day the amount of milk is increased by an ounce or two until, at the end of two weeks, if the calf has had no set-backs, it should receive about 12 per cent of its body weight, in milk, divided into three equal feedings in the 24 hours. To each feeding, one pint of lime water is added. We do not increase the milk above 12 per cent. Of course it is necessary to keep fairly close track of their gains in weight but we find

that one weighing a week will suffice for this purpose. The calf is kept muzzled and this method of feeding maintained for thirty days, after which time we are able to change gradually from whole milk to skim milk and allow the calf access to grain mixture and leafy hay. Calves under this method of handling will make some remarkable gains and are enough farther advanced and healthier to more than warrant the extra care necessary for the first month.

We find that another prime necessity for the health of young calves is that they be kept warm and free from drafts for the first few months. A shivering calf will not respond to the best of other care.

If calves develop scours—sometimes they do under the best of management—we withhold all raw milk, and sometimes substitute small quantities of boiled milk, or oatmeal, or barley gruel with lime water. No hard and fast rule can be laid down for the handling of these cases, as each presents its own particular problem. In this connection, however, I will say that if a calf develops scours or shows a marked decrease in vitality and resistance, we have injected 40 to 60 cc of its dam's fresh whole blood, subcutaneously, with very beneficial results. This may be repeated in 24 and 48 hours if necessary.

In conclusion let me say that only a few of the problems have been mentioned, which constantly confront the veterinarian in these dairy herds. A few methods of meeting some of these problems are presented, which have been somewhat helpful to us. If any of these will prove of benefit to men encountering similar situations, I will feel well repaid.

Discussion

Dr. D. H. Udall: Dr. Cushing has presented in a concise and comprehensive manner some important and interesting problems, not only in certified milk production but as applied to herds in general. I do not know that I can add anything to what he has said, but I will make a few comments from each of his sub-titles.

First, mastitis is one of the most important diseases of the cow. Often it is more destructive to a herd than either tuberculosis or abortion. The subject deserves investigation, not especially from a bacteriological standpoint, though that is important, but from a clinical standpoint. We need statistical tabulations on many cases of mastitis classified according to severity, and the type of infection, and arranged in groups with respect to the different methods of treatment.

The frequency of mastitis in certified herds, where every known precaution against infection is taken, leads one to wonder whether there is any adequate prevention. In some of the certified herds, the frequent exposure to moisture by washing the hind parts and the stall-beds, the clipping of the hind parts, and the frequent opening of outside doors in winter, is a reasonable explanation. Much more can be accomplished in the prevention than in the treatment of mastitis. Constant restraint in rigid stanchions, wet slippery floors, and

narrow stalls, all contribute to the disease. A bruised teat becomes infected and mastitis follows. Veterinary hygiene, in this respect, has not progressed in recent years. Dr. Cushing mentioned the influence of warm weather. From observation, and from conversation with herdsmen, I believe that mastitis does increase in very hot weather.

In the treatment of genital infections, pituitary extract has been mentioned. Doubt has been raised as to action of this preparation upon the uterus after the fetus has been expelled. During the past summer, Dr. Bardwell, of the Ambulatory Clinic, has conducted experiments that indicate a prompt action from the injection of pituitary extract after explusion of the fetus. Active debate always arises over the treatment of metritis, especially the acute form, as associated with retained placenta and other infections. Much of the argument for and against douching seems to rest on misunderstanding. To some, it means the free introduction of fluid in the hope that it may either wash out harmful debris, or act as a disinfectant. To others, it means the introduction of a limited amount of liquid in such a manner that its return with debris and pus is assured, no fluid remaining in the uterus.

In some herds, a suspension of a non-irritating disinfectant in a pint of oil is introduced into the uterus of each cow whenever metritis becomes prevalent, and in a few herds it is constantly used in a routine manner regardless of the presence of metritis. Each cow, on freshening, is treated. For this purpose one may use one-half ounce each of iodoform and bismuth subnitrate, or one-half to an ounce of bismuth formic iodid, in mineral oil. In regard to the value of this procedure there is also a difference of opinion; one group claims that it is of no value, while the other believes that it gives excellent results. In one respect it has a cardinal virtue: so far as I know, there is no claim that it does harm. In herds where it has been used for months or years, and the percentage of acute metritis and sterility remains low, it is not likely to be discontinued.

Dr. Cushing spoke of traumatic gastritis as a disease of considerable importance in commercial herds. In an average dairy practice, the deaths in a single year from this disease often exceed those from any other cause. For many years we have been making provisional or positive diagnosis of traumatic gastritis, but we have not accomplished much in the way of a cure. In the past year, Dr. Bardwell has devised a special rubber sleeve that simplifies the performance of rumenotomy, and transforms it into a clean surgical operation. Under these circumstances, there is no reason why exploration of the abdomen should not be practiced more often for the purpose of removing wires and nails when they begin to penetrate, and for the purpose of making a definite diagnosis. Under present conditions, there is no reason why this, an old operation, should not be more generally used. Its use brings increased skill in diagnosis.

PUBLICATIONS RECEIVED

The Register of Veterinary Surgeons. Royal College of Veterinary Surgeons, London, 1928. pp. 374.

Michigan State College of Agriculture and Applied Science, for the year 1927-1928, Catalog of the. East Lansing, Mich., 1928. pp. 336.

Intermountain Livestock Sanitary Association, Proceedings Annual Meeting. Ogden, Utah, January 10-11, 1928. W. D. Wright, Secretary, 312 Federal Bldg., Ogden, Utah. pp. 126.

Colorado Agricultural College Bulletin, Catalog, 1928-29. (Series 26, No. 9.) Fort Collins, Colo., Jan., 1928. p. 183.

Ontario Veterinary College, 1927, Report of the. Toronto, Ont., 1928. pp. 60.

Hemorrhagic Septicemia of Cattle. C. D. McGilvray. (Ontario Dept. of Agriculture Spec. Bul., March, 1928. pp. 4.)

Georgia State College of Agriculture, Catalog 1928-29. Athens, Ga., Mar., 1928. pp. 144.

Typical Medicinal Uses of the Carbon Arc. National Carbon Company, Inc., Cleveland, Ohio. pp. 22.

A NORMAL SHEEP URINALYSIS*

By DANIEL J. HEALY, JOHN F. BULLARD *and* HOWELL D. SPEARS

Kentucky Agricultural Experiment Station,
Lexington, Kentucky

A sheep urinalysis, any deviation from which would indicate an abnormal condition of the kidneys or of the organism, is desirable; in fact, it was found necessary during the study of a certain pathological condition in ewes and, failing to find such an urinalysis in the literature, it became necessary to establish one.

For the determination of such an uninalysis a 24-hour sample was not considered necessary and, therefore, the urine was collected between eight and ten o'clock each morning and examined at once.

As it was found necessary to obtain urine from the ewes with a catheter, an assistant held the ewe on her side with the hind legs forward; the external parts were washed with a weak antiseptic solution and, using the first two fingers of the left hand, the urethal orifice was exposed. A small, sterile, metal dog-catheter was passed through the urethra into the bladder, care being taken to insert the catheter into the dorsal portion of the urethral opening, as the ventral portion ends in a blind pouch. The urine was collected in a clean, 100-cc bottle and, as a rule, about thirty to fifty cubic centimeters of a concentrated urine were obtained.

The urinalysis was divided into three parts, clinical, chemical and microscopical. The clinical data included the date, the number and age of the ewe, whether or not pregnant, and the feed. The chemical data included the color, the clarity and the precipitate, which were observed while the urine stood in an urinometer glass; the specific gravity, which was determined with an urinometer, the reaction, which was tested with litmus; the percentages of urea, of ammonia, and of total nitrogen; the presence or not of albumen, sugar and acetone. The microscopical data included the formed elements thrown down in the centrifuge.

Analyses of the urine of forty ewes, from six to eight years old, were made during the months of October and November. Twenty-nine of these ewes were pregnant, eight were barren, and

*Presented at the sixty-fourth annual meeting of the American Veterinary Medical Association, Philadelphia, Pa., September 13-16, 1927.

with three the condition was doubtful. The ewes were kept in three pens. In the first pen they received whole corn and timothy hay, in the second, whole corn, timothy hay and salt, and in the third, whole corn, soybean hay and salt. This variation in feed made no noticeable difference in the composition of the urine.

For the urea, ammonia and total nitrogen determinations, if the specific gravity was more than 1.020, the urine was diluted one in one by volume with distilled water; if more than 1.030, it was diluted one in two, and if more than 1.040, it was diluted one in three. Hufner's method was used to estimate the urea. In

TABLE I—*Urea by the urease and the hypobromite methods*

NUMBER	UREASE METHOD %	HUFNER METHOD %	INCREASED READING %
451	3.84	4.20	0.36
452	0.43	0.45	0.02
453	2.50	2.00	-0.50*
454	2.16	2.60	0.44
459	3.36	4.30	0.94
461	4.17	4.50	0.33
462	0.32	0.65	0.33
463	3.00	4.60	1.60
466	3.45	3.95	0.50
467	2.40	3.20	0.80
469	5.53	5.80	0.27
471	0.60	0.70	0.10
472	4.92	4.90	-0.02*
473	3.20	4.20	1.00
475	5.40	6.20	0.80
476	2.82	3.20	0.38
478	1.94	2.40	0.46
484	0.55	1.80	1.25
486	1.70	2.24	0.54
489	2.50	3.00	0.50
		Average	0.51

*Diminished reading.

this method urea is decomposed by a strongly alkaline solution of sodium hypobromite, with the evolution of nitrogen and carbon dioxid, the nitrogen being collected and measured while the carbon dioxid is absorbed by the strongly alkaline solution. The solution is prepared as follows: 108 grams of caustic soda are dissolved in 250 cc of water, the solution cooled and 25 cc of bromin slowly added.

A Doremus ureometer is filled with the above solution and 1 cc of the urine, properly diluted if necessary, is carefully introduced; the apparatus is placed at room temperature and the reading made the following day.

The Hufner method gives readings which are somewhat high, probably due to decomposition of nitrogenous constituents other than urea, yet, for clinical purposes, it is most convenient and, therefore, twenty determinations were checked by the urease method, which is based upon the hydrolysis of urea to ammonium carbonate by the ferment urease. The method is applied as follows: 5 cc of urine and 100 cc of distilled water are placed in each of two 200-cc flasks, and to one is added 1 cc of a 10 per cent solution of urease.* The flasks are corked and placed at room temperature over night, or at 37° C. for three hours. Using a few drops of a one per cent alizarin solution as indicator, the contents of each flask are now titrated with decinormal hydrochloric acid. The difference in cubic centimeters between the two titrations multiplied by 0.06 gives the percentage of urea in the urine. Table I shows the results obtained by each method.

When using the Hufner method for urea, it should be remembered that the average of a number of readings is about one-half per cent higher than by the urease method.

Ammonia is present in fresh urine in the form of salts. If the urine is made neutral and tenth-normal caustic soda is added in the presence of formalin, free ammonia is formed and reacts as fast as formed with the formalin, forming hexamethyltetramine, thus:

$$4\ NH_3 + 6\ H_2CO \rightarrow N_4(CH_2)_6 + 6\ H_2O$$

The titration is done in the presence of neutral potassium oxalate, which prevents the obscuring effect of ammonia on the sharpness of the endpoint with phenolphthalein.

The following solutions are required:

1—A tenth-normal sodium hydroxid.

2—Commercial formalin, diluted one-half with tap water, and made neutral with sodium hydroxid, using phenolphthalein as the indicator.

This method was used as follows: About 60 cc of the urine, diluted if necessary, was treated with three grams of dry basic lead acetate, well stirred and filtered. The filtrate was treated with two grams of dry neutral potassium oxalate and filtered. Ten cc of the clear filtrate was diluted with 40 cc of distilled water, two or three grams of dry neutral potassium oxalate and a few drops of phenolphthalein solution added. The mixture was either acid or alkaline. If acid, it was neutralized with tenth-

*25-mg. tablets of urease may be obtained from Hynson, Westcott and Dunning, Baltimore. One tablet crushed in 5 cc of distilled water is used for each test.

normal sodium hydroxid. If alkaline, a few drops of dilute sulphuric acid were added and the mixture neutralized. Twenty cc of the neutral formalin solution was added, the mixture well stirred and again titrated with tenth-normal sodium hydroxid. The reading of the second titration represented the number of cubic centimeters of tenth-normal ammonia in 10 cc of the urine.

The total nitrogen was determined by the Kjeldahl method. Ten cc of urine, diluted if necessary, was placed in a Kjeldahl flask, 10 grams of powered sodium sulphate, 20 cc of concentrated sulphuric acid, and 1 cc of a saturated solution of copper sulphate added. The contents of the flask were boiled over a free flame for two and one-half hours. After cooling, 200 cc of tap water, saturated caustic soda solution sufficient to make slightly alkaline, and a small quantity of granulated zinc to prevent bumping were added; the ammonia was then distilled into a known quantity of tenth-normal sulphuric acid, and the acid titrated with tenth-normal caustic soda solution, using alizarin as the indicator. The difference in cubic centimeters between the known quantity of acid and the tenth-normal caustic solution required to neutralize, multiplied by 0.014, and by the dilution, if necessary, gives the percentage of nitrogen in the urine.

The senior writer,[1] while working with urine of the cow, found that the difference between the urea and ammonia nitrogen, and the total nitrogen calculated as hippuric acid, was a fairly accurate measure of the quantity present. This we found was not true with sheep urine and the distribution of the organic nitrogenous material will require further study.

Purdy's heat test for albumen, Rothera's test for acetone, and Fehling's copper sulphate method for sugar were used. In Purdy's test a small test-tube is filled two-thirds with clear urine and about one-sixth of this quantity of saturated sodium chlorid solution and five to ten drops of fifty per cent acetic acid are added. The contents of the tube are mixed and the upper third boiled. A white cloud in the heated portion shows the presence of albumen. In Rothera's test a gram of ammonium sulphate is dissolved in five or ten cc of urine in a test-tube, three drops of concentrated sodium nitroprussid solution are added and about one cc of strong ammonia is carefully floated over the mixture. A permanganate-colored ring shows the presence of acetone. This is a beautiful reaction and we have found it the most satisfactory test for acetone.

Upon examining the urine of forty ewes, we found the color varied from pale to deep yellow and, with two exceptions which were slightly cloudy, the urines were clear. The specific gravity varied from 1.010 to 1.052 with an average of 1.031. Thirty-eight of the urines were alkaline and two were acid. The urea varied from 0.32 to 5.53 per cent, with an average of 2.54 per cent; the ammonia, from none to 0.0385 per cent, with an average of 0.01 per cent; and the total nitrogen, from 0.24 to 3.10 per cent, with an average of 1.48 per cent. There was no sugar nor acetone in the urines and, with the exception of three showing a trace of albumen and one showing distinct albumen, there was no albumen present.

For the microscopical data 15 cc of the urine was precipitated in the centrifuge at 600 r. p. m. for ten minutes. The precipitate was examined on a microscope slide under a cover-glass with a magnification of 140 diameters.

Upon microscopical examination, epithelial cells and amorphous urates were found in all urines, a few red and white blood-cells were found in 70 per cent of urines, probably due to the method of obtaining the urine. Spermatozoa were found in two urines, *Trichomonas vaginalis* in four, bacteria in four, and yeast cells in one. Hippuric acid crystals were found in four urines, calcium oxalate crystals in two, and undetermined crystals in fifteen.

The urine of a normal sheep would, therefore, present an analysis approximately as follows:

Color, yellow; condition, clear; specific gravity, 1.031; reaction, alkaline; urea, 2.5 per cent; ammonia, 0.01 per cent; total nitrogen, 1.4 per cent. No sugar, no albumen and no acetone. Microscopical examination: epithelial cells, amorphous urates and, frequently, crystals, among them occasionally hippuric acid and calcium oxalate crystals; very occasionally, spermatozoa and parasites.

REFERENCE

[1]Healy, Daniel J.: The normal clinical urinalysis of the dairy cow Amer. Vet. Rev., xlii (1912), pp. 184-191.

PLANS FOR MINNEAPOLIS

The place: Minneapolis, Minnesota.
The time: August 7-8-9-10, 1928.
Headquarters: New Nicollet Hotel.
Official route: Burlington-Great Northern.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

CYSTIC CALCULI

By GERRY B. SCHNELLE, *Boston, Mass.*,

Angell Memorial Hospital

The accompanying photograph (fig. 1) shows a rather unusual X-ray plate of cystic calculi in a Boston terrier, female, ten years old.

At the time the picture was taken she had shown symptoms for six months, gradually increasing, but even then had no pain or hematuria, merely frequent urination. The owners did not

FIG. 1. Cystic calculi in a bitch.

wish to risk an operation and six months later had the dog destroyed. At that time she had become very emaciated but was still in good spirits and apparently did not suffer.

The close examination of the plate shows a marbled appearance and a thickening of the bladder-wall to about one-half inch.

The bitch had been fed almost exclusively on dog biscuits, with some table scraps to bolster the diet. The observation of similar cases seems to indicate that the trouble appears in dogs fed exclusively on cooked foods and that once operated, a diet of orange-juice, milk and raw meat prevents a recurrence of the calculi.

THE TREATMENT OF PERIODIC OPHTHALMIA WITH MERCURIC IODID*

By MAJOR R. A. KELSER, V. C.

U. S. Army Medical Department Research Board, Manila, P. I.

Several months ago the writer was asked to see a native pony which was found to be suffering from a typical case of periodic ophthalmia. The animal was seen also by Drs. Buencamino and Alagar, local veterinarians, who readily concurred in the diagnosis.

Having in mind the excellent results we have obtained with intravenous injections of red iodid of mercury in the treatment of epizootic lymphangitis, it occurred to me that this chemical might also prove of value in periodic ophthalmia. I therefore gave this animal, which weighed about 450 pounds, three intravenous injections of the iodid of mercury.

The doses were administered at 48-hour intervals and consisted of 0.30 gm. of the chemical suspended in 30 cc of distilled water. In preparing the mercuric iodid suspension we carried out the following procedure: The proper amount of drug is carefully weighed, placed in a sterile Erlenmeyer flask and 30 cc of sterile distilled water added. The flask is then vigorously shaken, in order to make as fine a suspension of the chemical as possible. Before it has a chance to settle, the mixture is poured into a 30-cc Luer syringe and promptly injected into the jugular vein. Leaving the needle in the vein, the flask and syringe are washed with 30 cc of normal saline solution and this is injected so that the animal gets the full dose of the iodid. Great care must be exercised to insure that none of the drug is injected into the vessel-wall or surrounding tissues.

Forty-eight hours after the first injection of the mercuric iodid the pony in question showed a marked improvement, which progressed with the two subsequent injections. The animal made a complete and rapid recovery and so far there has been no recurrence of the condition.

This is the only case we have had an opportunity to treat and obviously it would be the height of folly to draw definite conclusions from this single incident. However, the same is reported with the hope that others will try the treatment and report results obtained.

*Published with permission of the Surgeon General, U. S. Army, who is not responsible for any opinion expressed or conclusions reached herein.

The 0.30-gm. dose administered to the pony we treated is three-fifths of the daily dose of red iodid of mercury we have employed in the treatment of epizootic lymphangitis in American horses and mules. Thus, in the treatment of periodic ophthalmia in American horses I would recommend 0.50-gm. doses of the mercuric iodid given intravenously at 48-hour intervals. While three doses appeared to give results with the pony, as many as eight or ten injections can be safely administered.

In our lymphangitis work we are now using equal parts of red iodid of mercury and potassium iodid (0.50 gm. of each) in 30 cc of distilled water. This gives a solution of the double iodid of mercury and potassium and is more readily administered than the suspension of mercuric iodid alone. We have not employed this mixture over a sufficient period of time to determine whether or not it is as satisfactory as the mercuric iodid alone, but there appears to be no reason why it should not be.

RESULTS OF ANTI-RABIC VACCINATION

By Ashe Lockhart, *Kansas City, Mo.*

During the latter part of 1926 and the early part of 1927, the occurrence of rabies in Kansas City, Missouri, reached proportions which the Board of Health considered alarming. In the early months of 1927, the Kansas City Board of Health was authorized, by ordinance, to supervise the vaccination of all dogs in the City. About 20,000 were vaccinated during the first half of the year. While this has not completely stopped the occurrence of rabies, the disease occurs much more rarely than formerly.

According to the records of the Board of Health, during the first six months of 1927, 158 heads were examined in the laboratories of the General Hospital, 72 being positive cases of rabies. During the last six months of 1927, 52 heads were examined and no positive cases were found. During the first three months of 1928, three heads were examined and no positive cases were found. While this is perhaps not an accurate indication of the number of cases of rabies occurring in the City, it undoubtedly shows the diminished occurrence of rabies, and that the universal vaccination of dogs is so valuable that it behooves every community to make a practice of compulsory vaccination against rabies.

FREAK CALF

By H. A. McIntyre, *Maquoketa, Iowa.*

The accompanying photograph shows a half-blood Shorthorn calf, born to my Jersey cow, March, 15, 1928. The calf was

Fig. 1. Calf with three legs and no tail.

perfectly healthy at birth, but had only three legs and no tail. The calf weighed 135 pounds when shipped for veal at three weeks of age.

STRONGYLIDOSIS IN HORSES*

By Guy S. Jones, *Lohrville, Iowa.*

The cases which I wish to report seem to be unusual, either in the extent of the damage done by the parasites incriminated, or in the age of the animals damaged. During the last fall and early winter, I was impressed by the number of mature horses suffering more or less severely from infestation with strongyles, or what is more commonly termed, "palisade" worms. Upon the approach of severe cold weather, no less than a half-dozen

*Presented at the fortieth annual meeting of the Iowa Veterinary Medical Association, Des Moines, January 17-19, 1928.

mares suckling summer colts were found down, either in the barn or by straw-stacks, unable to rise, and died in a short time, due to the combined drain on their vitality by the colts, the parasites and the severe cold. But my attention was especially directed to one herd of fifty young and mature horses and mules.

On November 21, I was called to autopsy a suckling colt, which had been found dead in the stalk-field. The animal was in fair condition, and had seemed to be in good health. The abdominal cavity was filled with a bloody serum, and the appearance of the viscera indicated general peritonitis. The direct cause of this we easily located as an abscess, about two inches in diameter, in the mesentery. This had ruptured and emptied its contents into the abdominal cavity.

Upon opening the digestive tract, we found in the stomach several hundred larvae of the bot flies attached to the mucous membrane. The duodenum was literally occluded with a mass of *Ascaris equi;* a mass such as is frequently found in the duodenum of unthrifty pigs. And in the cecum and large colon were great numbers of the larvae and mature strongyles or palisade worms. These latter we then assumed to be the real cause of death—a septic embolism giving rise to the abscess, with the resultant peritonitis.

No attempt was made to differentiate the various species of this family. Hutyra and Marek classify four different species. Dr. E. A. Benbrook informs me that at the College, they have identified 53 different members of this group.

This owner has a permanent creek and timber pasture where he runs the young and spare horses. A casual examination of all animals that had been on this pasture showed many very thin, and the parasites could be identified in the droppings of any of them.

From the text-books one gets the impression that it is a disease affecting mostly the young animals, which had been my previous experience. But here was a group of 50 young and middle-aged horses and mules, many of the mature animals showing marked emaciation and weakness. One team of big six- and seven-year-old mules looked like a bunch of scare-crows, in spite of the fact that the bunch had had access to a good fall pasture and two weeks in a fresh stalk-field. Two three-year-old fillies were showing severe enteritis, with a profuse watery diarrhea. Ordinary medicinal doses had no effect in checking the diarrhea and the animals died the third day.

All animals were given 45 grains of sodium cacodylate intravenously and an aloes bolus, followed with a vermifuge powder in the feed for two weeks, a treatment which had been effective in previous experience.

At the beginning of the second week a three-year-old Percheron mare, that had raised a colt, developed enteritis with profuse diarrhea. This animal was given more than the usual dose of tannic acid with sulphocarbolates, and three ounces of tincture of opium per day during two days, but this failed to check the diarrhea, and she died the third day after showing evidence of enteritis.

The fourth animal to develop these symptoms, also a three-year-old, was given, every three hours, an ounce capsule of tannic acid and 240 grains of sulphocarbolates. Tincture of opium was given with this in varying doses, according to the symptoms, but over a 5-day period, a total of one pint was given with only temporary relief, and this animal went the way of the others from exhaustion and intoxication.

Observation of the herd, together with autopsies available, proved that the vermifuge powder was failing entirely to eliminate the parasites. To avoid a mistaken diagnosis, the Veterinary Division was asked to investigate. Dr. C. H. Covault and Dr. H. E. Beister were kind enough to brave the subzero weather and from their clinical and autopsy findings they were able to confirm the previous diagnosis. Consequently the treatment was changed to carbon tetrachlorid in 25- to 50-cc doses as recommended by Dr. Hall, after fasting 36 hours.

We selected carbon tetrachlorid in preference to oil of chenopodium because it is less toxic and, because the animals were unbroken, we did not fancy passing the stomach-tube on all that group to give raw linseed oil, as recommended with the latter drug. This treatment gave visible evidence of its efficacy from the second day on. A noteworthy feature is that no toxicity or ill effects whatever were noticed, even in the most emaciated subjects; also no more cases of severe enteritis have developed in the intervening thirty days, so that I, for one, am willing to give credit to carbon tetrachlorid as a very successful treatment for strongyle infestation. However, we still find a few parasites being passed off by some of the animals. Most of the others are still throwing off eggs, even a few which were given two doses at a ten-day interval. Otherwise the herd is making very satisfactory progress.

If one dose of carbon tetrachlorid is not sufficient, and ten days is too short an interval to repeat, both of which suspicions are borne out in this case, we are wondering just what would be the proper time to repeat this product, and would welcome any suggestions.

NEW GROWTH FOLLOWING CALK WOUND

By J. F. SHIGLEY, *State College, Pa.*

Subject: Black mare, white markings; age, 20 years.

History: This mare had a bad calk wound on the left hind foot about two years ago. The injury was very painful, although it did not cut through the coronary band or injure the wall of the hoof. For the first ten days weight was borne on the other legs. Healing was slow and granulation tissue was evident. The

FIG. 1. Growth following calk wound.

owner tried burning this tissue off, using actual cautery. After a time a lump appeared (owner's version) that did not bleed easily. This was not molested and developed into the growth shown in figure 1.

The owner used this mare all of the year except when the snow was deep. As she could not travel well on soft ground, she was

used for work either on the wagon or the plow when the footing was solid.

The mare was destroyed shortly after the picture was taken. No attempt was made to remove the growth or to section it after destruction of the mare.

ORCHITIS IN A BULL

By F. P. MATHEWS *and* G. H. ROBERTS

Department of Veterinary Science, Purdue University Agricultural Experiment Station, Lafayette, Indiana

Subject: A pure-bred Hereford bull, four years old.

History: Early in June, the animal was manifesting pain in the right testicle and the owner suspected a contusion of some sort. The pain subsided but the testicle increased in size,

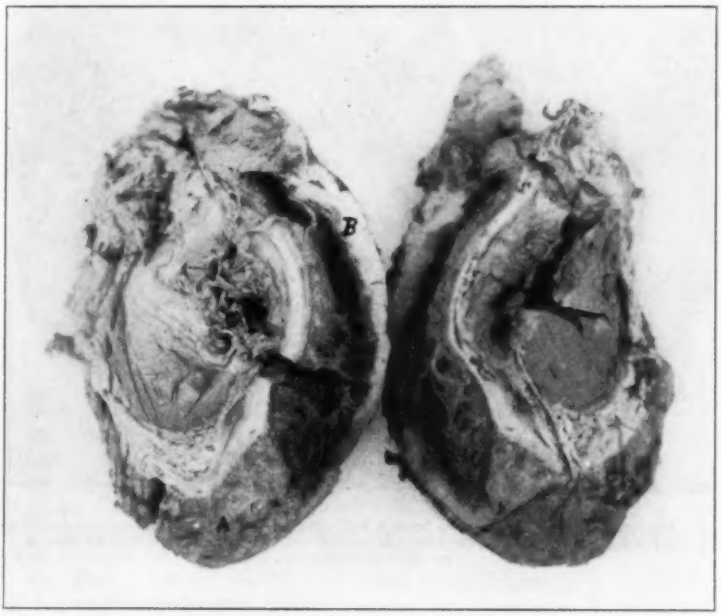


FIG. 1. A case of orchitis in a bull, showing (A) proliferative changes, (B) thickened tunica vaginalis and (C) a cavity containing necrotic tissue.

until about the middle of August, when it became an impediment to locomotion. The attending veterinarian diagnosed orchitis and tapped the organ at the owner's demand. About a pint of clear, straw-colored fluid was liberated from within the testicle.

The size of the testicle increased rapidly and was tapped again, ten days later, without results. Between the dates of the first and second trocarizations, the animal had lost 110 pounds, and it was decided to remove the diseased testicle. The operation was performed and the patient died the following day. The intact testicle was presented at the laboratory for examination.

Gross examination: The testicle measured 9 x 6 inches and weighed 2500 grams (5.5 lbs.). A longitudinal section showed

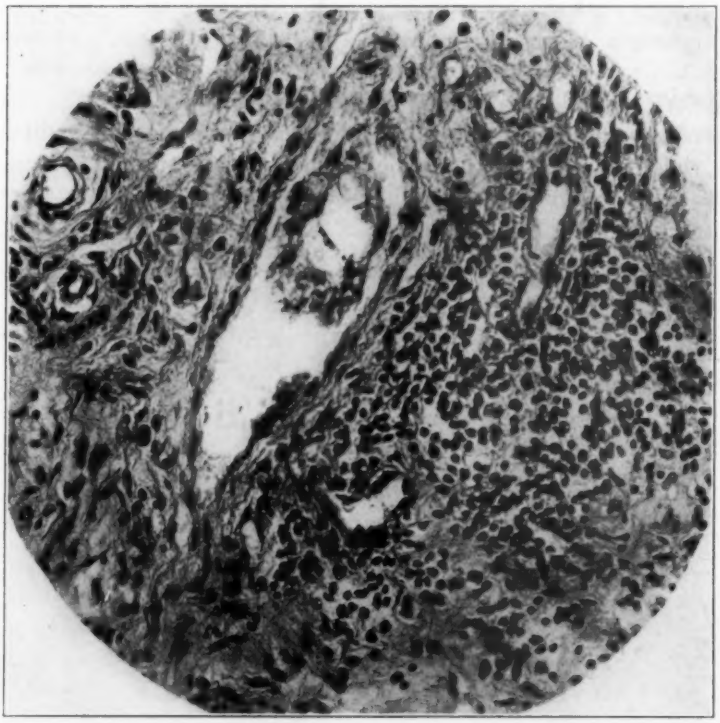


FIG. 2. Photomicrograph of a section from a bull's testicle, showing an atrophied convoluted seminiferous tubule, with its degenerated epithelium, marked fibrosis, and an area of round-cell infiltration.

an atrophied and fibrosed parenchyma. A cavity, lined by a dense fibrous wall, containing considerable necrotic tissue, occupied about one-third of the space previously occupied by the parenchyma. Along the border of the testicle, adjacent to the epididymis, there were proliferative changes which involved the tunica vaginalis, epididymis, and tunica albuginea. These three structures and the newly-formed tissue constituted a fibrous

band which varied one-half to three inches in thickness. The tunica vaginalis on the opposite side of the testicle was five-eighths of an inch thick and separated from the other structures by a band of necrotic and edematous tissue which contained much serosanguineous fluid.

Microscopical examination: The convoluted seminiferous tubules were distorted and much reduced in size. The epithelium for the most part consisted of a single layer of cells which showed various stages of degeneration. There was no evidence of spermatogenesis. The distortion of the tubules was the result of fibroblastic and angioblastic proliferation, which had evidently been in progress for some time, and greatly increased the amount of supporting tissue. The same type of proliferative changes, but evidently of a more recent occurrence, was responsible for the thickening of the tunica vaginalis. Focal accumulations of lymphoid cells were scattered throughout the newly-formed tissue. Purulent exudation and active granulation were present along the inner surface of the wall supporting the cavity found in the testicle.

Diagnosis: Fibroid atrophy of the convoluted seminiferous tubules was the diagnosis made. The extent of the fibrosis and the accumulation of lymphoid cells suggest *Bact. abortus* infection of long standing. The purulent process evidently followed the introduction of pyogenic bacteria at the time of trocarization.

BUREAU TRANSFERS

Dr. John L. Myers (Colo. '24), from Lincoln, Nebr., to South St. Paul, Minn., on meat inspection.

Dr. Leo P. Sullivan (St. Jos. '20), South St. Paul, Minn., to South St. Joseph, Mo., on meat inspection.

Dr. Clarence B. Henderson (K. C. V. C. '09), from Topeka, Kans., to Leavenworth, Kans, on meat inspection.

Dr. Chas. F. Pinkham (K. C. V. C. '09), from Leavenworth, Kans., to Topeka, Kans., on meat inspection.

Dr. Daniel J. Bynacker (U. S. C. V. S. '13), from Baton Rouge, La., to New Orleans, La.

Dr. Earl J. Meixel (K. C. V. C. '08), from Columbia, S. C., to Jackson, Miss., on tick eradication.

Dr. Francis D. Houston (O. S. U. '15), from Sioux City, Iowa, to Chicago, Ill., on meat inspection.

Dr. Allen A. Hiett, from Sioux City, Iowa., to Cincinnati, Ohio, on meat inspection.

Dr. Wm. A. Cornell (K. C. V. C. '17), from Austin, Minn., to Sioux City, Iowa, on meat inspection.

Dr. Roswell A. Telford (St. Jos. '17), from Chicago, Ill., to Waterloo, Iowa, on meat inspection.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Captain Lloyd C. Ewen is relieved from duty at the quartermaster depot, Fort Reno, Oklahoma, and directed to proceed at the proper time to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about August 10, 1928, for the Canal Zone for duty.

Captain Burlin C. Bridges is relieved from duty at Fort Bliss, Texas, effective on or about August 15, 1928, and directed to proceed to Fort Riley, Kansas, for duty.

Captain Frank M. Lee, Colorado Springs, Colo., who is under orders to report for duty at Fort Jay, N. Y., has been granted an extension of leave of 1 month and 3 days, and the date of his sailing from San Francisco has been deferred from March 21, 1928, to April 24, 1928.

Reserve Corps

New Acceptances

Bourne, Richard Franklin	Captain	312 E. Myrtle St., Fort Collins, Colo.
Bryan, Arthur Herbert	1st Lt.	4400 Raspe Ave., Baltimore, Md.
Evenson, Geo. Ernest	Captain	West Salem, Wis.
Fanslau, Charles Edward	Captain	22 E. Kinney St., Newark, N. J.
Gordon, Wm. Dane	Captain	Morristown, Ind.
Hamilton, Fred Carlisle	2nd Lt.	Kentland, Ind.
Jasme, Henry A.	1st Lt.	P. O. Box 88, Jacksonville, Fla.
Reading, Claude H.	2nd Lt.	Hopkins, Mich.

Separations

Bratten, Fred W.	1st Lt.	Declined reappointment.
Clarke, Fred Arthur	1st Lt.	O. R. C. status terminated, 2-14-28.
Gerety, Jos. Patrick	1st Lt.	Appointment terminated, 2-22-28.
Jasme, Henry A.	Captain	Discharged.
James, Thos. D.	Major	Appointment terminated, 3-8-28.
Marshall, Clarence J.	Lt. Col.	Transferred to Aux.-Reserves.
Burriss, Theo. L.	2nd Lt.	Appointment terminated, 3-8-28.
Young, James D.	2nd Lt.	Failed to accept reappointment.

Promotions

Mahan, Albert Ray, 614 G Street, Marysville, Calif., promoted to 1st Lieut.
Ward, Calvin A., 4161 Drexel Blvd., Chicago, Ill., promoted to 1st Lieut.

EXAMINATION FOR APPOINTMENT IN THE VETERINARY CORPS, REGULAR ARMY

Examination for the purpose of qualifying candidates for appointment in the Veterinary Corps, Regular Army, will be held beginning June 18, 1928, throughout the continental United States.

The applicant must be a male citizen of the United States, between the ages of 21 and 29 9–12 years, and a graduate of a recognized veterinary college.

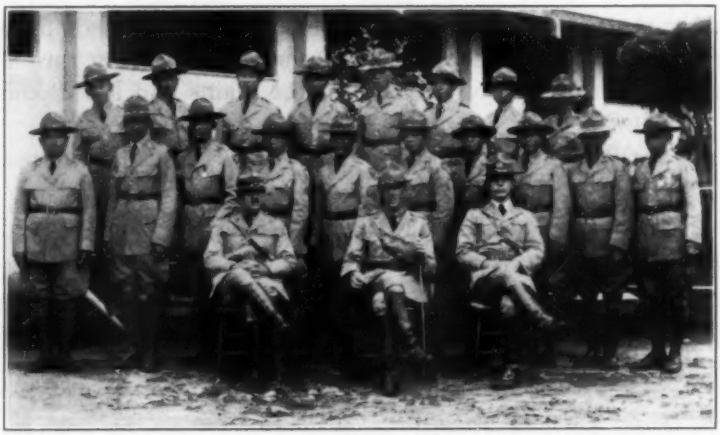
Application blanks, Form No. 62, A. G. O., may be obtained from The Adjutant General or from The Surgeon General, U. S.

Army, Washington, D. C., or from the Commanding Officer or Surgeon of any military post or station, and when completed should be forwarded direct to The Adjutant General, U. S. Army, Washington, D. C.

Any veterinarian desiring to take the examination should obtain the necessary application blank and submit it at the earliest practicable date.

VETERINARY ACTIVITIES AT CAMP STOTSENBURG, PHILIPPINE ISLANDS

Major Harold E. Egan, Veterinary Corps, United States Army, kindly sent the accompanying photograph from Camp Stotsenburg, Pampanga, Philippine Islands. It shows the Veterinary Detachment at that post, consisting of nineteen Philippine Scouts. The majority of these men have had years



Veterinary Detachment, Camp Stotsenburg, P. I.

of service in the veterinary branch and are well qualified in their technical duties, according to Major Egan. The animal population of Camp Stotsenburg is around 2400, which naturally makes it a very active place from a veterinary standpoint. The officers in the picture, from left to right, are: Captain Harry L. Watson, V. C.; Major Harold E. Egan, V. C.; and Lieut. E. Schweizer, V. C.

COMMUNICATIONS

LOCO DISEASE

To the Editor:

In December, 1892, I published in Bulletin 35, Kansas Experiment Station, "Some Observations upon Loco," and in the conclusions it is stated: "A careful survey of the experiments performed and observations noted leads me to the opinion that the disease known as 'loco' is the result of malnutrition or gradual starvation, caused by animals eating the plants known as 'loco weeds,' either *Astragalus mollissimus* or *Oxytropis lamberti*."

At that time we knew practically nothing about nutritional diseases and vitamins were unheard of. In these latter days, when literature, both scientific and lay, is so filled with nutritional discussions and alphabetical vitamins, I am wondering why some investigators do not resurrect good old loco disease and revivify it with some vitamins or mineral elements. Maybe the animals in the "short grass country" are now so well fed during the winter they do not have to hunt loco weeds for an existence. Will not some modern nutritional expert turn his machine-gun on this old "bogy" of the plains and let us know the results?

North Chicago, Ill., March 5, 1928. N. S. Mayo.

LIKES THE JOURNAL

To the Editor:

Sure appreciate your printing the proceedings of the United States Live Stock Sanitary Association in the Journal. Keep it up. Like the Journal fine.

W. H. Hilts.

Elko, Nev., April 11, 1928.

MISSOURI VALLEY MEETING POSTPONED

Dr. E. R. Steel, secretary of the Missouri Valley Veterinary Association, has authorized the announcement that the meeting of the M. V. V. A., originally planned to be held in Kansas City, in July, has been postponed. The meeting will be held, in all probability, during the week of the American Royal Live Stock Show (about the middle of November), in Kansas City.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Financial Report—Dr. M. Jacob, Treasurer
December 31, 1927

Bank balances, December 31, 1926		$ 3,614.97
Receipts, January 1, 1927, to December 31, 1927, as per statement		35,556.61
		39,171.58
Expenditures, same period, as per statement		32,269.31
		6,902.27
Placed on time deposit: Journal Fund	$1,372.66	
Relief Fund (interest)	40.58	
Schmidt Memorial Fund	330.00	
		1,743.24
Amount of cash in checking accounts, December 31, 1927		5,159.03
Revolving fund in hands of Dr. Hoskins	$ 500.00	
Loan to Relief Fund	100.00	
		600.00
Amount of cash, exclusive of time deposits		5,759.03
Cash on time deposit, December 31, 1926	$13,602.65	
Additional during period, as shown above	1,413.24	
Total amount now on time deposit		15,015.89
Total cash resources		20,774.92
Bonds (purchase price)		24,935.20
Total assets, December 31, 1927		45,710.12
Total assets, December 31, 1926		43,703.72
Gain for period		$ 2,006.40

Distribution of Assets

Fund	Cash	Bonds (Cost)	Time Deposits	Totals
A. V. M. A.	$1,129.88	$13,445.93		$14,575.81
Journal	4,629.15	9,509.01	$14,372.66	28,510.82
Relief		1,980.26	643.23	2,623.49
Totals	$5,759.03	$24,935.20	$15,015.89	$45,710.12

A. V. M. A. Fund

Bank Balance, December 31, 1926	$ 1,241.42	
Less old checks cashed	214.97	
		$ 1,026.45
Revolving fund in hands of Dr. Hoskins (see 1926 report)		500.00
Loan to Relief Fund (see 1926 report)		100.00
Receipts during period (as per statement)		9,460.60
Total		$11,087.05
Expenditures during period (as per statement)		10,627.17
Balance		459.88

Transfer to Schmidt Memorial Fund		330.00
Balance		129.88
Loan from Journal Fund		1,000.00
Balance		1,129.88
Loan to Relief Fund	$100.00	
Revolving fund in hands of Dr. Hoskins	500.00	
		$ 600.00
Balance, December 31, 1927		$ 529.88
Actual bank balance, December 31, 1927	$815.57	
Less checks not cashed	285.69	
Balance	$529.88	

JOURNAL FUND

Bank balance, December 31, 1926	$ 3,318.12	
Less old checks cashed	729.60	
		$ 2,588.52
Receipts during period (as per statement)		26,055.43
Total		28,643.95
Expenditures during period (as per statement)		21,642.14
Balance		7,001.81
Placed on time deposit		1,372.66
Balance, December 31, 1927		5,629.15
Loan to A. V. M. A. Fund*		1,000.00
Balance		4,629.15
Actual bank balance, December 31, 1927	$ 5,361.33	
Less checks not cashed	732.18	
Balance	$ 4,629.15	

SCHMIDT MEMORIAL FUND

Amount on time deposit, December 31, 1926		$ 365.25
Received from Secretary	$ 330.00	
Interest on time deposit	24.07	
		354.07
Total amount of fund, December 31, 1927		$ 719.32

SALMON MEMORIAL FUND

Amount on time deposit, December 31, 1926		$ 5,373.76
Liberty bond (face value)		500.00
		$ 5,873.76
Receipts:		
Interest on time deposit	$ 225.54	
Interest on bond	21.25	246.79
Total amount of fund, December 31, 1927		$ 6,120.55

*$1000.00 bond sold for Journal Fund, November 16, 1927, and proceeds placed to credit of A. V. M. A. Fund as a loan. $1000.00 transferred from Journal Fund to A. V. M. A. Fund, October 6, 1926, has not been returned. A. V. M. A. Fund now owes Journal Fund $2000.00.

Relief Fund

Amount on time deposit, December 31, 1926	$ 602.65
Interest during period	40.58
Total cash	$ 643.23

Bond Recapitulation

Fund	Par	Cost
A. V. M. A.	$14,000.00	$13,445.93
Journal	10,000.00	9,509.01
Relief	2,000.00	1,980.26
	$26,000.00	$24,935.20

Receipts from Secretary

January 1 to December 31, 1927

Monthly Receipts—Dr. H. P. Hoskins (A. V. M. A. and Journal Fund)

1927		
Jan. 20		$ 4,146.74
Feb. 17		4,234.41
Mar. 17		3,759.83
Apr. 17		1,738.77
May 20		2,494.27
June 17		1,598.23
July 18		1,484.73
Aug. 17		1,544.59
Sept. 25		1,705.46
Oct. 18		1,409.73
Nov. 16		3,585.97
Dec. 17		5,148.10
Nov. 16	Second Liberty Loan bond redeemed (Journal Fund)	1,000.00

Interest

Aug. 19	Bonds—A. V. M. A. Fund	$ 404.97
Aug. 19	Time deposits—Journal Fund	372.66
Aug. 19	Bonds—Journal Fund	404.96
Dec. 31—Bonds—A. V. M. A. Fund		127.54
Dec. 31	Bonds—Journal Fund	127.53
Dec. 31	Time deposits—Journal Fund	51.62
Nov. 16	Bonds—A. V. M. A. Fund	10.63
Nov. 16	Bonds—Journal Fund	10.62
Nov. 16	Time deposits—Journal Fund	154.67
Aug. 19	Time deposits—Relief Fund	32.14
Dec. 19	Time deposits—Relief Fund	8.44
Total Receipts		$35,556.61

Disbursements

January 1 to December 31, 1926

A. V. M. A. Fund

1927			
January	14	Dr. H. P. Hoskins, postage	$ 69.38
January	14	Schiffer Printing Co., stationery	62.25
January	14	Book Building, Inc., rent for January	118.00
January	14	Hazen, Lindsay & Carroll, premium on Treasurer's bond	198.33
January	14	Friesema Bros. Printing Co,, reprints	25.25
January	14	Lyford M. Moore, premium on Secretary's bond	25.00
January	29	Dr. H. P. Hoskins, miscellaneous petty expenses	28.00
January	29	Horse Association of America, dues	50.00
January	29	T. A. Falconnier, preparing Treasurer's report	20.00
February	1	Dr. T. A. Sigler, expenses attending meetings	86.02

February	1	Underwood Typewriter Co., typewriter	72.50
February	1	Una Anderson, salary for January	120.00
February	1	Dr. H. P. Hoskins, salary for January	416.67
February	12	Dr. T. A. Sigler, expenses attending Wis. Vet. Med. Asso. meeting	41.79
February	12	A. H. Donero, Inc., 1927 Hoskins Medal	23.50
February	12	Rand Kardex Service Corp., book case	39.29
February	12	Dr. H. P. Hoskins, expenses attending meetings	62.81
February	28	Dr. T. A. Sigler, expenses attending Univ. of Ill. Vet. Conference	20.59
February	28	Dr. B. T. Simms, expenses inspecting State Coll. of Wash.	43.56
February	28	Una Anderson, salary for February	120.00
February	28	Standard Mailing Machine Co., envelope sealer	40.00
March	17	Dr. H. P. Hoskins, postage	71.35
March	17	Book Building, Inc., rent for March	118.00
March	28	Dr. H. P. Hoskins, salary for March	416.67
March	28	Una Anderson, salary for March	120.00
April	7	Dr. T. A. Sigler, expenses attending O. S. U. Conference for Veterinarians	23.68
April	13	Dr. H. P. Hoskins, miscellaneous petty expenses	17.69
April	25	Dr. H. P. Hoskins, expenses attending meetings	73.97
April	25	Standard Bindery, Inc., binding "Reviews"	37.60
April	30	K. C. V. C. Alumni Asso., advertising	10.00
April	30	Una Anderson, salary for April	120.00
May	13	Schiffer Printing Co., stationery	32.10
May	13	Book Building, Inc., rent for May	118.00
May	28	Dr. H. P. Hoskins, postage	57.04
May	30	Dr. H. P. Hoskins, salary for May	416.67
May	30	Una Anderson, salary for May	120.00
June	13	Friesema Bros. Printing Co., membership directory	375.00
June	13	K. C. V. C. Alumni Asso., advertising	10.00
June	13	Jewel Emblem & Mfg. Co., radiator plates	68.00
June	13	Schiffer Printing Co., stationery	67.40
June	21	Dr. H. P. Hoskins, miscellaneous petty expenses	39.70
June	21	Dr. Earl F. Graves, 1st prize in essay contest	50.00
June	21	Dr. Wm. Edward Welsh, 2nd prize in essay contest	25.00
June	21	Rand Kardex Service Corp., safe	77.00
July	2	Una Anderson, salary for June	120.00
July	6	National Association of B. A. I. Vet., contribution for legal expenses	750.00
July	6	Book Building, Inc., rent for July	118.00
July	6	Schiffer Printing Co., stationery	24.25
July	26	Dr. T. A. Sigler, expenses attending meetings of Mo. Valley, N. Dak. and Minn. Vet. Med. Associations	112.57
July	26	Dr. H. P. Hoskins, postage	37.73
July	26	Dr. H. P. Hoskins, salary for July	416.67
July	26	Una Anderson, salary for July	120.00
August	13	Dr. H. P. Hoskins, postage	35.36
August	30	Una Anderson, salary for August	120.00
August	30	Dictaphone Sales Corp., dictaphone	275.00
August	30	Schiffer Printing Co., stationery	29.00
September	2	Dr. H. P. Hoskins, expenses attending meeting	26.86
September	2	Frank McElroy, auditing records of Secretary	37.50
September	5	Book Building, Inc., rent for September	118.00
September	5	Dr. H. P. Hoskins, miscellaneous petty expenses	27.19
September	5	T. A. Falconnier, preparing report of Treasurer	5.00
September	5	Schiffer Printing Co., stationery	27.25
September	26	Friesema Bros. Printing Co., reprints	42.50
September	26	K. C. V. C. Alumni Asso., advertising	10.00
September	26	Richmond & Backus Co., office supplies	53.00
September	26	Dr. H. P. Hoskins, salary for September	416.67

September 26	Una Anderson, salary for September	120.00
September 26	Prof. Dr. B. Bang, honorarium, 64th annual meeting A. V. M. A.	250.00
September 26	Una Anderson, expenses attending convention	93.61
September 26	Dr. H. P. Hoskins, expenses attending convention	103.36
October 11	Journal of the A. V. M. A., subscriptions for honorary members	111.00
October 11	Dr. T. A. Sigler, expenses attending convention	93.52
October 11	E. S. Fish, typing	10.64
October 11	Dr. H. P. Hoskins, postage	109.82
October 11	Schiffer Printing Co., printing	135.00
October 22	Dr. M. Jacob, expenses attending convention	89.63
October 22	Dr. J. P. Turner, typing	3.00
October 22	Adelphia Reporting Bureau, reporting convention	250.20
October 29	Una Anderson, salary for October	120.00
November 14	Dr. H. B. Cox, convention expenses	323.65
November 14	Book Building, Inc., rent for November	118.00
November 14	Schiffer Printing Co., stationery	129.51
November 14	The Wolf Detroit Envelope Co., envelopes	38.65
November 25	Master Reporting Co., reporting convention	287.25
November 25	Dr. H. P. Hoskins, miscellaneous petty expenses	17.46
November 25	Dr. H. P. Hoskins, postage	75.42
November 25	Dr. H. P. Hoskins, salary for November	416.67
November 25	Una Anderson, salary for November	120.00
December 12	Dr. N. S. Mayo, expenses visiting A. V. M. A. office	28.26
December 12	Dr. H. P. Hoskins, expenses attending meetings	46.87
December 12	Dr. Reuben Hilty, expenses attending meetings	55.68
December 12	K. C. V. C. Alumni Asso., advertising	10.00
December 12	Jewel Emblem & Mfg. Co., radiator and lapel emblems	113.92
December 12	Dr. F. S. Jones, expenses attending meeting of Comm. Vet. Biologics	81.37
December 12	Una Anderson, salary for December	120.00
December 12	Dr. Reuben Hilty, expenses attending Nebr. St. Vet. Med. Asso. Meeting	84.32
Total		$10,627.17

DISBURSEMENTS

January 1 to December 31, 1927

JOURNAL FUND

January	14	Dr. H. P. Hoskins, postage	$ 32.58
January	29	Friesema Bros. Printing Co., printing January Journal	955.75
February	1	Underwood Typewriter Co., typewriter	107.50
February	1	Dr. H. P. Hoskins, miscellaneous petty expenses	63.19
February	1	Alice Laverick, salary for January	140.00
February	12	Dr. H. P. Hoskins, miscellaneous petty expenses	46.63
February	12	Dr. H. P. Hoskins, postage	70.02
February	12	Friesema Bros. Printing Co., printing February Journal	956.00
February	12	Rand Kardex Service Corp., book case	35.16
February	12	Book Building, Inc., rent for February	118.00
February	12	Friesema Bros, Printing Co., reprints	118.41
February	28	Everton Engraving Co., halftones	78.35
February	28	Wolf Detroit Envelope Co., envelopes	143.28
February	28	Alice Laverick, salary for February	140.00
February	28	Dr. H. P. Hoskins, salary for February	500.00
March	28	Friesema Bros. Printing Co., reprints	170.95
March	28	Alice Laverick, salary for March	140.00
March	28	Friesema Bros. Printing Co., printing March Journal	1,800.00
April	7	Everton Engraving Co., halftones	63.55
April	7	Book Building, Inc., rent for April	118.00
April	13	Friesema Bros. Printing Co., printing April Journal	960.00
April	13	Dr. H. P. Hoskins, miscellaneous petty expenses	40.68

April	13	Dr. H. P. Hoskins, postage	63.74
April	25	Dr. H. P. Hoskins, postage	79.63
April	26	Standard Bindery, Inc., binding Journal A. V. M. A.	27.60
April	30	Dr. H. P. Hoskins, salary for April	500.00
April	30	Alice Laverick, salary for April	140.00
April	30	Friesema Bros. Printing Co., reprints	450.48
May	13	Everton Engraving Co., halftones	102.37
May	13	Dr. H. P. Hoskins, back numbers of Journal	38.70
May	28	Dr. H. P. Hoskins, postage	47.72
May	28	Friesema Bros. Printing Co., printing May Journal	969.75
May	30	Friesema Bros. Printing Co., reprints	85.25
May	30	Alice Laverick, salary for May	140.00
June	13	Book Building, Inc., rent for June	118.00
June	13	Friesema Bros. Printing Co., printing June Journal	968.62
June	21	Dr. H. P. Hoskins, postage	53.34
June	21	Dr. H. P. Hoskins, miscellaneous petty expenses	77.63
July	2	Dr. H. P. Hoskins, salary for June	500.00
July	2	Alice Laverick, salary for June	140.00
July	2	Friesema Bros. Printing Co., reprints	178.83
July	6	Everton Engraving Co., halftones	117.98
July	26	Friesema Bros. Printing Co., printing July Journal	974.75
July	26	Dr. H. P. Hoskins, postage	52.21
July	26	Alice Laverick, salary for July	140.00
August	15	Friesema Bros. Printing Co., printing August Journal	971.37
August	15	Book Building, Inc., rent for August	118.00
August	15	Friesema Bros. Printing Co., reprints	163.96
August	15	Everton Engraving Co., halftones	84.79
August	15	Friesema Bros. Printing Co., reprints	73.37
August	15	Dr. H. P. Hoskins, postage	56.91
August	30	Dr. H. P. Hoskins, salary for August	500.00
August	30	Alice Laverick, salary for August	140.00
August	30	Dictaphone Sales Corp., dictaphone	197.20
August	30	Schiffer Printing Co., envelopes	15.00
September	2	Dr. H. P. Hoskins, miscellaneous petty expenses	31.52
September	26	Friesema Bros. Printing Co., printing Sept. Journal	985.00
September	26	Alice Laverick, salary for September	140.00
September	26	Dr. H. P. Hoskins, postage	59.06
October	11	Friesema Bros. Printing Co., reprints	51.76
October	11	Book Building, Inc., rent for October	118.00
October	11	Dr. H. P. Hoskins, miscellaneous petty expenses	63.46
October	11	Friesema Bros. Printing Co., printing October Journal	980.12
October	22	Dr. H. P. Hoskins, postage	49.74
October	29	Dr. H. P. Hoskins, salary for October	500.00
October	29	Alice Laverick, salary for October	140.00
November	14	Friesema Bros. Printing Co., reprints	119.63
November	14	Dr. H. P. Hoskins, miscellaneous expenses	65.60
November	14	Dr. H. P. Hoskins, postage	56.21
November	25	Friesema Bros. Printing Co., printing Nov. Journal	985.00
November	25	Dr. H. P. Hoskins, back numbers of Journal	28.45
November	25	Alice Laverick, salary for November	140.00
December	12	Book Building, Inc., rent for December	118.00
December	12	Friesema Bros. Printing Co., reprints	80.85
December	12	Dr. H. P. Hoskins, postage	38.37
December	12	Friesema Bros. Printing Co., printing Dec. Journal	986.87
December	12	Dr. H. P. Hoskins, miscellaneous petty expenses	87.07
December	28	Everton Engraving Co., halftones	92.18
December	28	Dr. H. P. Hoskins, salary for December	500.00
December	28	Alice Laverick, salary for December	140.00
		Total	$21,642.14

ASSOCIATION MEETINGS

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The forty-fourth annual meeting of the Veterinary Medical Association of New Jersey was held at New Brunswick, January 24-25, 1927, as guests of the College of Agriculture. Dr. J. H. McNeil presided.

Dr. H. E. Bemis, of the Veterinary School, University of Pennsylvania, gave a very interesting paper on "Some Fundamentals Requisite in Sterility Work." Dr. Bemis stressed the importance of a proper fundamental knowledge for this work, careful diagnosis and treatment. Dr. Earle B. Hopper, of New York City, led the discussion of this paper.

Dr. R. R. Birch, of the New York State Veterinary College at Cornell University, talked on "Bang Abortion Disease, Its Nature and Handling." Dr. Birch's talk was supplemented with lantern-slides showing some very interesting data on agglutination tests conducted over a period of several years. The discussion was opened by Dr. M. F. Barnes, of the Pennsylvania Bureau of Animal Industry.

Dr. T. E. Munce, Director, Pennsylvania Bureau of Animal Industry, presented a paper on "The Assistance Private Veterinarians Can Render in the Control of Contagious Abortion." His paper showed the important rôle played by the private practitioner in abortion disease control and the necessity of adopting systematic measures for the control of this condition.

A banquet was held, the evening of the first day, Dr. Wm. Herbert Lowe, of Paterson, presiding as toastmaster. The following speakers addressed the gathering:

Dr. J. G. Lipman, Director, New Jersey Agricultural Experiment Station.
Professor Franser Metzger, Rutgers University.
Hon. W. B. Duryee, Secretary of Agriculture, Trenton, N. J.
Dr. J. H. McNeil, Chief, Bureau of Animal Industry, Trenton, N. J.
Dr. H. E. Bemis, Professor of Veterinary Surgery, Veterinary School, University of Pennsylvania.
Dr. R. R. Birch, Professor-in-Charge of Experiment Station, New York State Veterinary College, Cornell University.

After a short business session, the second day's program was opened with a most interesting paper by Dr. M. Dorset, Chief, Biochemic Division, U. S. Bureau of Animal Industry, on "Hog Cholera and Allied Diseases, Their Differentiation and Control."

Dr. Dorset emphasized the fact that the "basis of control measures should be sanitation and good hygiene, coupled with the very prompt use of anti-hog cholera serum, unless we can definitely exclude hog cholera." The discussion was opened by Dr. E. R. Cushing, of New Brunswick.

Dr. J. M. Hendrickson, in charge of poultry disease work at the New York State Institute of Applied Agriculture, Farmingdale, Long Island, presented a paper on poultry diseases, which was discussed by Dr. F. R. Beaudette, Poultry Pathologist, New Jersey Agricultural Experiment Station, New Brunswick.

The program for the afternoon consisted of two papers. The first was "Some Phases of Canine Practice," by Dr. Frank E. McClelland, of Buffalo, N. Y. The speaker stressed the necessity for strict adherence to ethical standards in the profession and the promotion of good feeling between individual neighboring practitioners. Dr. John B. Hopper, of Ridgewood, and Dr. J. Payne Lowe, of Passaic, discussed Dr. McClelland's paper. The second paper, "Some Problems of the Agriculture of Tomorrow," was ably presented by Professor F. G. Helyar, College of Agriculture, New Brunswick.

The following officers were elected for the ensuing year: Dr. J. H. McNeil, Trenton, president; Dr. H. C. Millar, Asbury Park, 1st vice-president; Dr. R. W. Butterworth, Paterson, 2nd vice-president; Dr. H. Ticehurst, Tenafly, treasurer; Dr. E. R. Cushing, New Brunswick, secretary.

There was a good attendance at the meeting and much interest was manifest.

E. R. CUSHING, *Secretary.*

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The fifth semi-annual meeting of the State Veterinary Medical Association of Texas was held at Forth Worth, January 23-24, 1928. Headquarters were at the Texas Hotel.

The visiting veterinarians were welcomed to Fort Worth by Dr. Holman Taylor, secretary of the Texas State Medical Association and editor of the *Texas State Journal of Medicine.* In his address Dr. Taylor paid the following tribute to the veterinary profession:

> Veterinarians, members of that branch of the medical profession which administers to the domestic animals which serve mankind as food, bearers of burdens, or friends, are important factors in bringing about better

health conditions among human beings and are saving the live stock producers millions of dollars by eliminating sickness among his herds. The veterinarian is a coworker of equal importance with the physician who administers to sick humanity, in maintaining the health of the community.

Dr. A. E. Flowers, of Dallas, responded to Dr. Taylor's address of welcome. He urged veterinarians to strive continually for higher standards and greater proficiency in their professional work.

Dr. C. C. Hisel, state veterinarian of Oklahoma, discussed anaplasmosis in cattle. This disease made its appearance in Oklahoma about seven years ago and since that time may have spread into Texas, although no cases have been definitely diagnosed, according to Dr. Hubert Schmidt, of Texas A. & M. College.

Dr. Scott A. Burnham, of Clarendon, then presented a paper on "Hydrops Ascites," a disease reported to be prevalent in young steers. This condition is commonly known as "waterbelly." Dr. Burnham stated that the treatment is purely symptomatic, until more is known concerning the disease.

Dr. T. O. Booth, laboratory director, Live Stock Sanitary Commission of Texas, interested his hearers with a well-worked-out plan of testing and applying sanitary methods in the care of poultry, from the standpoint of public health economics.

The evening of the first day, an elaborate and beautifully appointed banquet was served to about one hundred guests who were delightfully entertained by local celebrities with song and music. The speaker of the evening was Dr. J. C. Anderson, state health officer. Dr. M. E. Maier, of Orange, president of the Association, acted as toastmaster. Dr. R. E. Flowers, of Fort Worth, had charge of the affair and received much praise for providing such an enjoyable occasion.

The meeting Tuesday morning opened in the Medical Arts Building. Dr. T. C. Terrill, of the Terrill Laboratories, Fort Worth, gave a lecture on rabies, illustrated with lantern-slides. He gave the results of his experiments to determine the length of time necessary for killing rabies virus with various antiseptics. Dr. Terrill answered many questions relative to the incubation period of rabies in both man and animals, as well as concerning first-aid treatment. Dr. Terrill reported having administered 4200 anti-rabic treatments with only two fatalities.

Dr. H. O. VonRosenberg, first assistant state veterinarian, presented a paper entitled, "Bovine Tuberculin Testing in the Field," This paper contained a wealth of information in the

form of statistics showing losses to both man and beast, financial and otherwise, caused by the disease.

Dr. J. W. Williamson, chief meat and dairy inspector of Beaumont, outlined the work he is doing in the field of municipal milk and dairy inspection.

Dr. J. S. Grove, B. A. I. inspector-in-charge, Fort Worth, gave an interesting lecture on "Postmortem Lessons." He discussed the value of meat inspection to the public health, adulterations used by the packers, coloring matters used in the process of bleaching and other closely related facts. Dr. Grove commented on the shortage of veterinarians in the federal service and stressed the need for a larger number of qualified men entering the government service.

The members considered it a great privilege to be addressed by Dr. Livingston Anderson, laboratory director, Texas State Board of Health. His subject concerned cattle reacting to the tuberculin test. Dr. Anderson made the statement that the veterinarian and the physician constitute the only branch of medicine capable of making good sanitarians. He also related some interesting experiences in connection with his work, giving an example of a veterinarian who had condemned nine reacting cows. The Board of Health had called the veterinarian a fool for slaughtering the cows. Dr. Anderson then took specimens from no-lesion reactors and inoculated guinea pigs. He later showed the Board of Health that the test was 100 per cent accurate. Dr. Anderson advised the veterinarians not to be discouraged if they received reports from the packers that reactors had failed to show lesions in all cases. Dr. Anderson proved through his address that he is a real friend of the veterinarian.

The meeting ended with a luncheon at the Blue Cross Cat and Dog Hospital, given by the Globe Laboratories. A clinic was conducted by Drs. Peck and Blackburn.

It was the unanimous opinion of those present that this meeting was the best in the history of the Association. About one hundred members were in attendance. The Ladies' Auxiliary held a meeting on the first day. This was presided over by the President, Mrs. U. E. Marney, of San Antonio.

DEE PEARCE, *Secretary.*

MICHIGAN STATE COLLEGE POSTGRADUATE SHORT COURSE

The fifth annual postgraduate short course for veterinarians was held at Michigan State College, January 23-27, 1928.

Formerly, the programs have extended over a period of five days but this year, at the suggestion of some of those who have attended the courses, the regular program covered only three days: Tuesday, Wednesday and Thursday. Monday and Friday were given over to registration and consultation with individual members of the staff of the College. This change in the plan of the course seemed to be a success, as the attendance for the three days was very good, fifty-five being registered, and there seemed to be no lack or loss of enthusiasm at any time during the program. In fact, the number of men attending the program of the last day was as large as, if not larger than, that of any other day of the course. Sixty-seven were present at the banquet Thursday evening.

On Monday evening the Michigan State College Student Chapter of the A. V. M. A. furnished an entertaining and interesting program. Dr. Milton Shaw, of Lansing, gave an excellent talk on certain developments in human medicine and showed that his profession had come up to its present exalted position, from the obscure and lowly, through a process similar to that from which the veterinary profession is about to emerge.

Tuesday was designated as Cattle Day and Dr. E. T. Hallman was in charge. The following topics were discussed: A summary of our present knowledge of abortion, by Drs. I. F. Huddleson and A. S. Schlingman; sterility, causes, prevention and treatment, by Drs. E. T. Hallman and L. H. Smith; undulant fever and hypersensitiveness, by Drs. I. F. Huddleson and H. J. Stafseth; some problems that have arisen in connection with the tuberculosis eradication program, by Drs. T. S. Rich and D. W. Beck; the "anatomy of the microscope," by Dr. F. W. Chamberlain (practitioners now consider the microscope a part of standard equipment!); the bacteriology of the udder, by Mr. J. P. Torrey; bovine mammitis, by Dr. L. H. Smith; traumatic gastritis, by Dr. F. E. Stiles, of Battle Creek. Provisions were made for a laboratory section, at which Dr. Smith presented work on the reproductive organs of cattle and Drs. E. T. Hallman and L. B. Sholl gave a demonstration of the technic of semen examination.

In the evening, Dr. Schlingman, of Parke, Davis and Company, gave an interesting and instructive lecture, illustrated with moving pictures, on the manufacture of biological products. Professor Edwards presided over the discussion which followed.

Wednesday was devoted to poultry and miscellaneous topics with Dr. Stafseth in charge. The merits of the different tests for bacillary white diarrhea were discussed by Dr. Frank Thorp. A highly instructive paper on the control of bacillary white diarrhea in the field was presented by Dr. A. H. DeGroot, of Dundee. Dr. J. W. Patton gave an interesting talk on drugs, and his address was supplemented by a discussion of the autonomic nervous system and its reaction to certain drugs, by Dr. A. R. McLaughlin. Dr. Ward Giltner presented an interesting paper on "Pests, Whence, How and Whither," in which he described the struggle of man with disease and emphasized the importance and necessity of adequate veterinary education.

In the afternoon, Dr. M. J. Smead, of Parke, Davis and Company, gave an interesting account of his experience with bacterins and hygiene in the control of roup. It seems that bacterins are of considerable value in preventing and curing this troublesome ailment.

Poultry parasites and their treatment were ably discussed by Dr. W. L. Chandler, who emphasized the fact that there is only one vermicide, as far as worms of poultry are concerned, and that is iodin.

In a laboratory section, demonstrations were given on examination for tuberculosis, macro- and microscopic, microscopic examination for coccidia, fresh material and stained sections, and finally differential diagnosis of certain common poultry diseases that can be diagnosed by gross lesions. These demonstrations were given by Drs. E. P. Johnson, H. J. Stafseth and Frank Thorp.

In the evening a lively round-table discussion was presided over by Dr. B. J. Killham.

Thursday was Small-Animal Day and Dr. J. P. Hutton was in charge. Dr. E. K. Sales discussed the feeding of dogs and puppies and Dr. C. L. Bliss, State Toxicologist, and Mr. W. G. Geagle, State Analyst, gave very interesting and instructive talks on the possibilities and impossibilities of chemical analyses for poison. They stressed the importance of a careful local investigation of suspected cases of poisoning and the absolute necessity of collecting and submitting specimens for examination in a correct manner—*one that will hold in court*—at the same time supplying the chemist with a complete history of the case. Dr. Reuben Hilty, president of the A. V. M. A., gave an excellent address on rabies.

A demonstration of the best technic in the examination for internal parasites of small animals was given by Dr. Chandler. This demonstration was highly appreciated by the audience.

As has already been mentioned, the banquet was very well attended. H. H. Halladay, D. V. M. (Hon.), secretary of Michigan State College, pointed out the importance of veterinary service and education to the nation. Dr. H. Preston Hoskins, our new professor of A. V. M. A., gave an interesting and entertaining account of the almost innumerable activities of the national association. Dr. Reuben Hilty then took up the status of the veterinarian in the past and at the present time, putting a good deal of stress on the things with which he has to contend in his effort to make a success of his work.

On Friday, several veterinarians took advantage of the opportunity to consult with members of the staff regarding special lines of work, such as parasitology and milk hygiene.

As a whole the course was a great success. The contributions of practicing veterinarians were particularly noticeable and valuable. It is not long ago since it was almost impossible to get practitioners to appear on a program of this sort. This year the success of our program was due largely to the active participation on their part.

At a meeting of the Central Michigan Veterinary Medical Association, in December, the writer obtained some excellent suggestions for the short course program and, on behalf of those responsible for the course, takes this opportunity to express sincere appreciation to those present at that meeting for the assistance given in planning the course.

H. J. STAFSETH, *Reporter.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Gannett and Risley Hospital, 59 Lawrence St., Brooklyn, February 1, 1928.

Due to the absence of both the President and Vice-president, Dr. Robt. S. MacKellar presided. The attendance was very good, a very large number of non-members being present. The minutes of the December meeting were read and approved.

The first number on the program, "Discussion and Demonstration of Parasites," was opened by Dr. E. R. Cushing. The sub-

ject was a very interesting one, which was evident by the interest taken by the following members: Dr. Henry Amling presented "Parasites in Wild Animals." Dr. Geo. H. Berns discussed the use of carbon disulphid in the treatment of bots. Dr. M. L. Morris brought up the subject of the prevalence of whip worms. This was discussed by Drs. H. K. Miller, H. B. Risley, J. Elliott Crawford, Geo. J. Goubeaud and others.

Other discussions were entered into by Drs. MacKellar, Garbutt, Spevack, Hoog and Altman. Judging from the discussion, parasites seem to be a big problem.

The second part of the program, "Clinical and Surgical Cases," was left to the last, but we did not get to it, as the discussions and case reports ran the meeting quite late.

Dr. Goubeaud reported a very interesting case report, that of rubbing a dog with powdered sulphur to rid it of sand fleas. In rubbing the sulphur into the dog's hair it took fire and burned the dog and owner very badly. Quite a discussion followed this case report in explanation of the cause of the sulphur taking fire.

Dr. R. W. Gannett reported on the twentieth annual Conference for Veterinarians at Cornell University; Dr. E. R. Cushing, on the meeting of the Veterinary Medical Association of New Jersey; and Dr. R. S. MacKellar, on the meeting of the Long Island Veterinary Medical Association.

Under new business, Dr. MacKellar brought up for consideration the meeting of the New York State Veterinary Medical Society, which is to be held in the metropolitan district in June. He advised any member who had any arrangements in connection with same to start planning.

The discussion of "smoker or dinner" was laid on the table for further consideration.

Dr. Russell B. Booth, of New York City, was elected to membership.

C. P. Zepp, *Secretary.*

ALABAMA SHORT COURSE FOR GRADUATE VETERINARIANS

During the week of February 6, 1928, the fifth annual Short Course for Graduate Veterinarians was held at Auburn, Alabama, by the College of Veterinary Medicine, Alabama Polytechnic Institute. Dr. W. L. Boyd, of the University of Minnesota, gave the subject of sterility in cows a prominent place. As a rule it is a sequel of infectious abortion. The much-talked-of corpus

luteum was considered, with its normal and abnormal functions. Normally it is said to prevent estrum during pregnancy and possibly in some indirect way prevent abortion. Retention of the corpus luteum when the cow is not pregnant may prevent the regular and possibly irregular estrum, hence, it prevents the formation of graafian vesicles, ovulation and conception. Removal of the retained corpus luteum in some way brings about an estrual period and ovulation, and, with all other functions being normal, conception.

Removal of the corpus luteum in cows and heifers (not more than two months gone in pregnancy) will usually be followed by abortion. Some exceptional cases occur where the pregnant cows come in heat regularly or irregularly during the entire term of pregnancy. There are yet inexplicable or unknown actions of the corpus luteum. Ovarian cysts are the direct or indirect result of extension of infection and inflammation of the uterus. These cysts may be ruptured and quite often return. Maturation of ova rarely occurs after repeated formation of cysts, hence, ovarian cysts may mean temporary or permanent sterility.

Dr. Boyd described his manner of removing a corpus luteum from an ovary and also of rupturing an ovarian cyst. He also gave demonstrations in examining and disinfecting the cervix and canal of the os uteri. He also gave his methods of handling retained placenta and warned all about avoiding infection of the uterus and injury of the maternal cotyledons. He brought out the ways in which cows become infected with the abortion germ, and discussed indigestion, Johne's disease and the methods of making a diagnosis of pregnancy. Dr. Boyd's talks and demonstrations were very interesting and the practitioners were pleased and greatly benefited by his instruction.

Dr. Charles Murray, of Iowa State College, gave very profitable talks on hog cholera, hemorrhagic septicemia, infectious enteritis and swine flu. The differential characters, clinical symptoms and postmortem lesions were made clear and distinct. Dr. Murray stated that most of the books written on diseases of poultry were written for laymen, hence, many pathological errors had crept into poultry medical literature. Eventually these errors must and will be eliminated. Dr. Murray gave a new list of drugs for poultry, with minimum and maximum doses for each. He also discussed the common diseases, such as pox, infectious and nutritional roup, canker, fowl cholera, fowl

typhoid, white diarrhea, coccidiosis, etc. His lectures were very valuable to the practitioners who are now doing quite a good deal of poultry practice.

Dr. F. D. Patterson made a typical, practical autopsy on a hen. He also gave a good talk on feed, feeding, and the care of dogs. At another time he told how to handle distemper in dogs and gave the most recent developments in the research work on dog distemper.

Dr. Ed. Everett gave a demonstration of bleeding some sixty Rhode Island Reds, to get blood samples for making the agglutination test for white diarrhea. All the veterinarians in attendance were enabled to see all the steps, from bleeding to reading of the tubes at the end of the test.

Dr. J. M. Bryan described his method of treating parturient paresis with glucose injections into the circulation. Quite a spirited and extended discussion was held over the various methods of meat and milk inspection. It was quite generally conceded that state and city milk inspectors were often laymen who had no training or qualifications for doing efficient work. Let the medical men, too often in charge of meat and milk inspection, take notice that unqualified inspection will sooner or later discredit and ruin the work. Every meat and milk inspector should be a graduate veterinarian.

One evening was given over to educational moving-pictures. One film came from Oregon. It was the excellent and famous film on salmon poisoning, in which the action, life history and anatomy of the salmon fluke were vividly and accurately presented. Dr. B. T. Simms, one of the investigators and producers of the film, is a native of Alabama and is an Auburn graduate.

Dr. J. S. Koen demonstrated the characteristic lesions of hog cholera by the use of three virus-pig carcasses from the State Serum Plant.

The examination of veterinarians who desired to become approved for testing accredited herds was held here and quite a number of our practitioners stood the state and federal tests. The Alabama Veterinary Medical Association met in business session, on Friday, February 10. The following officers were elected for the ensuing year: President, Dr. J. M. Bryan, Birmingham, Ala.; vice-president, Dr. E. A. Davis, Columbus, Ga.; secretary-treasurer, Dr. C. A. Cary, Auburn, Ala.

C. A. CARY, *Dean.*

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Society convened at Albany, February 8, 1928, after a luncheon at Jack's Restaurant.

Among the veterinarians present was Dean Moore, of the New York State Veterinary College, who was called upon by President McCartney and gave a brief address referring to the increasing importance of the profession as a factor in improving live stock conditions, the increasing demands for better supervision over milk, meat and other animal food products, and the limited number of men taking up veterinary medicine. The speaker referred to the poultry industry as an example of the change in the demands upon the veterinarian for his services.

The speaker of the day, Dr. Paul B. Brooks, deputy commissioner of the State Health Department, then addressed the meeting, his subject being the "New State Milk Code." This code becomes effective July 1, 1928. The speaker discussed the more exacting requirements for the different grades of milk; the practical elimination of Grade B, except when produced by herds for which application has been made for an official tuberculin test; the importance of more careful physical examination by veterinary inspectors; and the disposition of rejected individuals.

An animated discussion then followed as to the details of barn and dairy inspection, the opportunity for the veterinarian in this work and the necessity of cooperation between the producer, the veterinarian and the authorities involved.

Six new members were admitted to the Society at this meeting and nearly fifty were in attendance.

J. G. WILLS, *Secretary.*

ONTARIO VETERINARY ASSOCIATION

The semi-annual meeting of the Ontario Veterinary Association was held on February 9, 1928, in the Prince George Hotel, Toronto. The meeting was well attended, the addresses and the papers read were of a high character and the discussions which took place were of a nature that indicated exceptional interest and knowledge regarding the subjects. The meeting was called to order at 11 a. m., by the President, Dr. R. A. McIntosh.

The first item on the program was an address by the President, in which he outlined the program for the activities of the

Association for the ensuing year and also pointed out the accomplishments of the profession in Canada since the profession had become active in the control and eradication of disease. Mention was made of the fact that there had never been sufficient recognition of the unique position Canada enjoyed in her freedom from many of the so-called animal plagues. Foot-and-mouth disease has never occurred in Canada. The same could be said of contagious pleuro-pneumonia, and also that the Health of Animals Branch has kept the country relatively free from hog cholera. Mention also was made of the progress that has been made in the eradication of tuberculosis, and also that the country was practically free from glanders and some of the other diseases of an infectious character.

Following this the minutes of the last meeting were read and a discussion on several items of business pertaining to the Association were dealt with. One of these items was the appointing of a delegation of the members of the Association to interview the Department of Agriculture at Ottawa in behalf of those members of the profession who are engaged in civil service work. The meeting then adjourned for lunch, after which papers were presented and discussed.

The first paper was given by Dr. L. A. Wilson, district supervisor of the Meat and Canned Foods Act, of Toronto, on the subject of "Meat Inspection for the General Practitioner." The paper was well received and considerable discussion referable to this subject followed. The next item was on the subject of "Blackleg," by Dr. S. R. McKelvey, a practitioner, of Beeton. The speaker mentioned that this disease was prevalent in his locality and he was able to give a very good clinical picture of the condition, and also outlined the proper method of vaccinating against the disease. Mention also was made of the treatment and cure of one case by the use of the anti-blackleg serum. Discussion followed regarding the most useful preventive inoculation to use in this disease and in summing up it was decided that the aggressin was probably the most beneficial.

Another paper, by Dr. R. MacDonald, of Richmond Hill, was given on "Typical and Atypical Cases of Milk Fever." This again was a subject that was followed by a good deal of discussion. Dr. R. G. Law, of the Government Experimental Fur Farm, at Kirkfield, spoke on certain phases of fox-ranching. The speaker, whose time is devoted entirely to this work, was able to outline certain definite measures in the control of diseases which

affect foxes and also as to proper measures which may be taken in the feeding, management and care of these animals. Dr. J. A. Campbell, of Toronto, discussed this paper at considerable length and also Dr. A. A. Kingscote, who had spent a summer with a veterinarian on Prince Edward Island. His discussion related principally to the proper handling of lungworms in foxes as followed by his preceptor on the Island.

Owing to lack of time, two other items, "Observations in Dairy Practice," by Dr. N. E. McEwen, and "Hemorrhagic Septicemia," by Dr. A. H. MacLeod, were omitted. The question-box was then opened and a lively discussion took place over the questions which had been presented. The final address of the afternoon was by Dr. C. D. McGilvray, principal of the Ontario Veterinary College, on "The Diversification of Veterinary Training and Practice." In his address the doctor dealt briefly with the history of the profession, showing how other branches of the work developed as the profession moved forward and advanced, until today the profession is a highly important organization in so far as the control and eradication of infectious disease is concerned and also in regard to food hygiene, dairy inspection, etc. Mention was also made of the fact that the training which the veterinary students receive nowadays enables them to be particularly useful as laboratory technicians in disease control work and diagnosis.

The meeting then adjourned and, at 6:30, a banquet having been arranged for, all of the members present took advantage of this period of time for social intercourse, at the conclusion of which Professor J. J. R. Macleod, of the University of Toronto, addressed the audience about the work of William Harvey, this being the tercentenary of the publication of his book on the circulation of the blood. The speaker was introduced by Dr. McGilvray and the address which followed was very interesting indeed. The speaker went back through the history of anatomy, pointing out the difficulties the ancients had in developing their work, bringing it up to the point where William Harvey finally discovered the circulation of the blood. This discovery, Professor McLeod intimated, was really the turning-point in the study of medicine and since that time great strides had been made. At the conclusion of his address the speaker was warmly applauded and by way of appreciation an honorary membership in the Association was conferred upon him.

All those present expressed themselves as being delighted with the meeting and felt that much benefit had been derived from the educative viewpoint.

R. A. McIntosh, *Res. Sec. for Ontario.*

MANITOBA VETERINARY ASSOCIATION

The annual meeting of the Manitoba Veterinary Association was held on February 10, 1928, at the Royal Alexandra Hotel, Winnipeg. About fifty members were in attendance, the chair being taken by the President, Dr. J. B. Still, Winnipeg.

The Secretary-Treasurer's report showed one hundred and fifteen active members and eight honorary members, while the balance sheet showed a balance of $715.55 standing to the credit of the Association, at the end of the year.

The following officers were elected for the ensuing year: Dr. W. A. Shoults, Winnipeg, president; Dr. Owen McGuirk, Dauphin, vice-president; Dr. William Hilton, Winnipeg, secretary-treasurer and registrar. Council: Dr. J. B. Still, Winnipeg; Dr. A. L. Alton, Portage la Prairie; Dr. J. R. Fisher, Brandon; Dr. Owen McGuirk, Dauphin; Dr. W. Hilton, Winnipeg; Dr. W. A. Shoults, Winnipeg; Dr. H. H. Ross, Brandon. Examining Board: Drs. J. B. Still, A. L. Alton and H. H. Ross. Auditors: Drs. M. T. Lewis and T. G. Sprague.

Dr. J. A. Stevenson, of Winnipeg, and Dr. D. Robinson, of Victoria, B. C., were elected honorary members of the Association.

In view of the success of the summer clinics held during the past two years, it was decided to hold a clinic this coming summer at Portage la Prairie.

The question of the practicing veterinarians being given a larger share of the work of tuberculin-testing being carried out under the policies of the Dominion Government, Health of Animals Branch, on passing a special examination as to their qualifications for such work, was discussed, and a committee was appointed to wait upon the Provincial Government and solicit their assistance in pressing this matter on the Dominion Government.

The following addresses and papers were delivered at the afternoon session:

Address: "Safeguarding the Public Milk Supply," by Dr. A. W. Shoults, Provincial Board of Health.

Paper: "The Use of the Stomach-Tube," by Dr. H. R. McEwen, Stonewall.

Address: "The Importance of Increasing Sheep Production," by Dr. A. L. Alton, Portage la Prairie.
Paper: "Losses Occurring Among Young Pigs," by Dr. T. G. Sprague, Health of Animals Branch, Winnipeg.
Paper: "Hemorrhagic Septicemia; Its Clinical Significance in Manitoba," by Dr. M. T. Lewis, Stonewall.
Address: "Operations Performed at 1927 Summer Clinic and Observations on Same," by Drs. H. H. Ross and J. R. Fisher.
Address: "The Present Condition of Live Stock Industry of the Province and the Part the Veterinary Surgeon Could Take in Promoting More and Better Live Stock," by Mr. J. H. Evans, Deputy Minister of Agriculture.

WM. HILTON, *Secretary.*

NORTHEASTERN PENNSYLVANIA VETERINARY MEDICAL CLUB

A meeting of the Northeastern Pennsylvania Veterinary Medical Club was held at the Terminal Hotel, Wilkes Barre, February 23, 1928. Eighteen veterinarians were in attendance. A business session was held at 11 o'clock, followed by a luncheon at 12 noon, after which the veterinarians motored out to the spacious and well-equipped veterinary hospital of the Lehigh and Wilkes Barre Coal Company, where a very interesting clinic was conducted by Dr. H. E. Bemis, professor of veterinary surgery, University of Pennsylvania. Dr. H. R. Church, deputy state veterinarian, also gave a very interesting talk.

T. D. JAMES, *Secretary.*

POLK COUNTY (IOWA) VETERINARY MEDICAL ASSOCIATION

Twenty-five veterinarians met at the Hotel Savery, Des Moines, Iowa, February 27, 1928, and organized the Polk County Veterinary Medical Association. Practically all fields of the veterinary profession were represented by the men in attendance, who took dinner together and then enjoyed a smoker during the evening.

Among those who contributed to the program were Drs. C. H. Stange and H. L. Foust, of Iowa State College, Ames; Dr. C. J. Scott, Knoxville, secretary of the Iowa State Veterinary Medical Association; Dr. J. I. Gibson, St. Joseph, Mo., former state veterinarian of Iowa; Dr. Peter Malcolm, state veterinarian; Dr. J. A. Barger, B. A. I. inspector-in-charge of tuberculosis eradication in Iowa; Dr. C. W. Deming, B. A. I. inspector-in-charge of meat inspection at Des Moines; Major J. H. Gould, and several others. All stressed the value of properly coordinated veterinary

activities, as well as the benefits to be derived from county associations.

Officers were elected as follows: Dr. F. F. Parker, Des Moines, president; Dr. C. C. Franks, Grimes, vice-president; Dr. A. H. Quin, Jr., Des Moines, secretary.

A. H. QUIN, JR., *Secretary.*

ROCK VALLEY VETERINARY MEDICAL ASSOCIATION

The veterinarians of Rock County, Wisconsin, held a meeting March 12, 1928, and organized an association to be known as the Rock Valley Veterinary Medical Association.

The meeting, which followed a dinner given by Dr. and Mrs. L. W. Cleland, of Janesville, was attended by every veterinarian in the County. The Association was formed with every member of the profession in the County as a charter member. The organization papers state the object of the Association is the advancement of veterinary science and to further the protection of the live stock industry of Rock River valley.

Dr. J. S. Koen, of Bloomington, Ill., was present as speaker of the evening and gave an interesting talk on "The Object and Results of Organization."

As soon as the Association is in good working order the veterinarians of adjoining counties will be invited to join.

The officers elected for the ensuing year are: Dr. L. W. Cleland, Janesville, president; Dr. A. B. Russell, Beloit, vice-president; Dr. A. J. Knilans, Janesville, secretary-treasurer.

ARTHUR J. KNILANS, *Secretary.*

ILLMO VETERINARY MEDICAL ASSOCIATION

The Illmo Veterinary Medical Association met in Dr. A. A. Moore's veterinary hospital, Edwardsville, Ill., March 22, 1928. It was a very interesting and instructive meeting. More than forty veterinarians attended. The program consisted of a clinic in the morning. Dr. H. R. Schwarze, of East St. Louis, Ill., conducted a poultry clinic on infectious diseases. Dr. W. R. Michael, of Highland, Ill., and Dr. A. A. Moore, of Edwardsville, Ill., demonstrated the pullorin test for bacillary white diarrhea. Dr. H. J. Schlesinger, of New Athens, Ill., demonstrated caudal anesthesia in a cow. Dr. A. H. Kyle, of Highland, Ill., gave a demonstration of spaying heifers. Dr. A. Darling, of St. Louis, Mo., gave a very interesting talk and demonstration on the use

of the bronchoscope in dogs. Dr. S. W. Haigler, of St. Louis, Mo., performed the sutureless spaying operation on a bitch. Dr. J. T. Jennemann, of St. Louis, Mo., gave a demonstration of tail amputation of dogs.

The literary program was presented in the afternoon. The president's address, by Dr. L. J. Miller of Waterloo, Ill., was presented and answered with an address of welcome by Mr. C. W. Perry, of the Chamber of Commerce of Edwardsville, Ill. Mr. Don Robinson, Chief, Bureau of Animal Industry, Illinois Department of Agriculture, gave a very interesting talk on the work that is being done on tuberculosis eradication in the state of Illinois. Dr. H. R. Schwarze, of East St. Louis, Ill., presented a paper on "Animal Diseases Transmissible to Man." Dr. H. J. Schlesinger, of New Athens, Ill., read a very practical paper on "Sterility in Cows." Dr. S. W. Haigler, of St. Louis, Mo., gave an interesting talk on diseases of small animals. The rest of the afternoon was taken up in discussing the above subjects. It was indeed gratifying to observe the interest the attending veterinarians took in the program. It was one of the outstanding meetings of the Illmo Veterinary Medical Association.

H. R. Schwarze, *Reporter.*

LONG ISLAND VETERINARY MEDICAL ASSOCIATION

The Long Island Veterinary Medical Association held its regular monthly meeting at the Brookville Country Club, Glen Cove, on March 1, 1928. Dr. Chas. S. Chase, of Bay Shore, gave a most excellent report on the Veterinary Conference held at Ithaca, in January. The meat and milk situation, as related to rural districts, was discussed at some length by the members present. Problems encountered in everyday practice were also brought up and discussed. The meeting was followed by a banquet.

J. M. Hendrickson, *Secretary.*

A PRIZE FOR SOMEONE

In an endeavor to check the mosaic disease of sugar-cane, which is one of the most destructive of tropical plant diseases, the State of Rio de Janeiro, Brazil, has offered a prize of approximately $1,200 to any scientist who, before the end of the current year, will ascertain its cause and determine an effective method for combating it.

—*Science.*

NECROLOGY

W. J. BUTLER

Dr. W. J. Butler, of Fond du Lac, Wisconsin, died, April 20, 1927. He was a graduate of the Ontario Veterinary College, class of 1889, and was a member of the Wisconsin Veterinary Medical Association. Although not a member of the A. V. M. A., he was a subscriber to the JOURNAL for many years.

HOMER PATRICK FLOWE

Dr. Homer P. Flowe, of Asheville, North Carolina, died August 17, 1927. He was born at Clear Creek, N. Car., May 8, 1889, spent two years at Lenoir College and then entered the United States College of Veterinary Surgeons, from which he was graduated in 1913. He was assistant state veterinarian of North Carolina from 1913 to 1917.

Dr. Flowe served in the Second Division, for 27 months, during the World War. He was in four major engagements: Argonne, Chateau Thierry, St. Mihiel and Meuse. In 1920 he was awarded a medal by the U. S. government, in recognition of his war record. He held a commission as Captain in the National Guard.

Dr. Flowe joined the A. V. M. A. in 1916.

FRANK H. AMES

Dr. Frank H. Ames, of Canton, Ill., died at his home, December 21, 1927, in his 71st year. The verdict of a coroner's jury was to the effect that death resulted from morphin poisoning, accidentally self-administered. He was a graduate of the Kansas City Veterinary College, class of 1896, and had been in practice at Table Grove and Canton, Ill., since graduation.

MOSES MOORE DEW

Dr. M. M. Dew, of Wilson, North Carolina, died suddenly, January 21, 1928. He was in partnership with Dr. L. J. Herring at the time of his death.

Born January 17, 1896, Dr. Dew attended the North Carolina State College of Agriculture and Engineering and received the degree of B. S. in 1920. His course of study was interrupted by

the World War. He saw eighteen months of active service, both in France and Germany, with rank of lieutenant, in the Quartermaster Corps. Following his graduation from North Carolina State College, Dr. Dew entered Ohio State University and received the degree of D. V. M. in 1922.

Dr. Dew was married, January 27, 1927, to Miss Ethel Warren, who survives him.

R. L. TRITTON

Dr. R. L. Tritton, of Bishop, California, died February 9, 1928. He was born in England, in 1849, and was registered in California as a graduate of the Ontario Veterinary College, class of 1899. (His name does not appear in the registry of alumni of that institution, maintained in the JOURNAL office. Editor.) Dr. Tritton, for the past few years, was the only veterinarian located in Inyo County, California. He is survived by his widow.

W. L. C.

SAMUEL V. RAMSEY

Dr. Samuel V. Ramsey died at his home in Waycross, Ga., March 10, 1928.

Born in New York City, February 9, 1860, Dr. Ramsey, while quite young, moved with his parents to Parkville, Ill., where he secured his early education. He attended the Chicago Veterinary College and was graduated in 1889. He then located at Tuscola, Ill., where he established a very fine practice. Later Dr. Ramsey went to Terre Haute, Ind., where he helped organize the Terre Haute Veterinary College, in 1909. He served as president and director, in addition to teaching the principles and practice of veterinary medicine and obstetrics.

Dr. Ramsey joined the A. V. M. A. in 1909. About twelve years ago, he removed to Waycross, Ga., and established a practice there. He is survived by his widow, three daughters and one son, Dr. S. V. Ramsey, Jr., of Terre Haute, Ind.

JOHN S. NICHOLAS

Dr. John S. Nicholas, of Charlottesville, Va., died at the University of Virginia Hospital, March 21, 1928. Death was due to peritonitis following an operation for appendicitis.

Born in Buckingham County, Virginia, April 1, 1889, Dr. Nicholas secured his early schooling in the county schools of

Buckingham and the Charlottesville (Va.) High School. He then entered the United States College of Veterinary Surgeons and was graduated in 1917.

During the World War, Dr. Nicholas served in the Veterinary Corps as a second lieutenant and saw service in France. After his discharge from military service he held a temporary appointment on the Virginia Live Stock Sanitary Board, in connection with tuberculosis eradication. During the session 1920-21, Dr. Nicholas was assistant professor of veterinary science at the Virginia Polytechnic Institute. He resigned this position to enter private practice at Charlottesville.

Dr. Nicholas joined the A. V. M. A. in 1919 and served as Resident Secretary for Virginia, 1925-1928. He was a member and past president of the Virginia State Veterinary Medical Association. He was one of the outstanding veterinarians of Virginia and was held in high esteem by all his colleagues.

He is survived by his widow, one daughter and twin sons.

W. G. C.

EDWARD RAFTER

Dr. Edward Rafter, of Hamburg, N. Y., passed away, March 22, 1928, after an illness of several weeks.

Born in North Evans, N. Y., August 31, 1872, Dr. Rafter attended the Hamburg (N. Y.) High School and later the Ontario Veterinary College, from which he was graduated in 1895. He practiced in Hamburg continuously up to the time of his fatal illness.

Dr. Rafter joined the A. V. M. A. in 1911. He was a member of the New York State Veterinary Medical Society and the Western New York Veterinary Medical Association. He also held membership in the Hamburg Business Men's Club and the Hamburg Chamber of Commerce. For the past ten years he was secretary of the Hamburg Grange.

Hamburg sincerely mourns the early death of a useful citizen, who had endeared himself to the community. Dr. Rafter is survived by his widow and one brother.

C. E. H.

RICHARD EBBITT

Dr. Richard Ebbitt, of Papillion, Nebraska, died in his room at the Zeeck Hotel, March 23, 1928, following a stroke of paralysis. He was seventy-two years of age and his death is believed

to have been hastened by his grief over being refused to be allowed to return to Ireland, his native land, to spend the rest of his days.

Educated in Edinburgh and a graduate of the Royal College of Veterinary Surgeons, Dr. Ebbitt came to America in 1883. He located in Omaha and was naturalized in 1888. For two years he was veterinary editor of the old weekly *World-Herald*. In 1893 he entered the service of the U. S. Bureau of Animal Industry and later the Quartermaster Corps of the U. S. Army. He saw service in the Philippines during the Moro uprising. Dr. Ebbitt went to Alaska during the Klondike gold rush of 1899-1900 and prospected at Teller. He returned to the United States and practiced at Grand Island, Nebr., until 1912, when his health failed and he returned to Ireland.

Dr. Ebbitt was banished from Ireland by the British authorities, in 1916, during the Sinn Fein uprising. His deportation became the subject of a diplomatic investigation at Washington. On February 14, he left Papillion to return to Ireland, where he said he wanted to die "because it was nearest heaven." The Irish Free State authorities stopped him at Queenstown and he was not allowed to step on Irish soil. He was taken to Germany and then returned to America, his spirit crushed.

Dr. Ebbitt joined the A. V. M. A. in 1892. He was a member of the Missouri Valley Veterinary Association. He is survived by a nephew.

E. L. GLACKEN

Dr. E. L. Glacken, of near Richwood, Ky., died March 29, 1928, at Booth Memorial Hospital, Covington, Ky., of blood poisoning. It is reported that Dr. Glacken cut one of his fingers while repairing a wire fence and the wound became infected.

Born at Richwood, January 2, 1867, Dr. Glacken attended common school and the Cincinnati Veterinary College. He received his diploma from the latter institution in 1913.

Dr. Glacken joined the A. V. M. A. in 1917. He was County Live Stock Inspector for Boone County, Kentucky.

BERT C. ELDREDGE

Dr. Bert C. Eldredge, of Swanton, Ohio, died at his home, March 30, 1928, of rabies. The infection is believed to have been contracted from a dog treated by Dr. Eldredge about six months previously, although he took the Pasteur treatment subsequently.

Born in Tedrow, Ohio, October 19, 1870, Dr. Eldredge attended the public schools and Fayette Normal. He then entered the Ontario Veterinary College and was graduated in 1902. He immediately located in Swanton and continued in practice there up until his death.

Dr. Eldredge attended the veterinary conference at Ohio State University, March 21-23, and filed an application for membership in the A. V. M. A. while in Columbus. He was a member of the Ohio State Veterinary Medical Association and the Northwestern Ohio Veterinary Medical Association. He served as a member of the Council of Swanton for several years. He is survived by his widow and two daughters.

GUSTAVUS H. GRAPP

Dr. Gustavus H. Grapp, chief field veterinarian of the Live Stock Sanitary Section of the Maryland State Board of Agriculture, died at his home in Baltimore, Md., March 31, 1928. His heart had been giving him some trouble for the past two or three years. The last few months and, in fact, up to the last day, he attended to his duties in his usual efficient and cheerful manner. Heart failure was responsible for his sudden death.

Born in Stetten, Germany, May 6, 1866, Dr. Grapp received his early education in that country, coming to America when yet a boy. While still a young man, he became the superintendent of the Union Transfer Company, being stationed in Washington and Baltimore. He held this position for twenty-two years. He was married to Elida H. James, on June 14, 1892.

Entering the United States College of Veterinary Surgeons, Washington, D. C., in 1904, Dr. Grapp was graduated in 1907. Upon finishing school he entered the federal service as a dairy inspector and was stationed at Frederick, Md. During the three years he held this position, his activities were practically in a pioneer field. To such men as him and his associates should be given much of the credit for the present condition of dairy inspection on the Washington milk-shed.

Upon resigning this position, Dr. Grapp established himself in private practice at Port Deposit, Maryland. Here his energy and ability again asserted themselves. He built up a most lucrative practice, which was maintained for a period of four years. In 1917 he left this practice to accept a position with the Mary-

land State Board of Agriculture, which position he held until his death.

It would indeed be very difficult to estimate how much Dr. Grapp has done for the District of Columbia and the state of Maryland. During his entire career he was outstanding for his professional ability and his insistence for accuracy to the last detail. Even with this latter trait, he was always generous in dealing with others.

Dr. Grapp joined the A. V. M. A. in 1915, and attended many of the meetings. He was a charter member of the Maryland State Veterinary Medical Association, in which he was very active, serving two terms on the Executive Committee. He was also a member of the Maryland State Veterinary Examining Board. He was affiliated with the following Masonic organizations: Waverly Lodge No. 152, F. & A. M.; Druid Royal Arch Chapter No. 28; Jerusalem Council No. 2; Royal and Select Masters; Monumental Commandery No. 3; Knight Templar; Boumi Temple (Shrine) and Scimitar Club of Boumi Temple.

Dr. Grapp is survived by his widow and an adopted daughter.

E. M. P.

HONORÉ F. LAINÉ

Dr. Honoré F. Lainé, a prominent Cuban veterinarian died recently. Dr. Lainé belonged to a distinguished Cuban family and had held important government positions. He was a member of the faculty of the National Veterinary College of the University of Havana. Dr. Lainé had attended one of the American veterinary colleges and because of this he suffered persecution from Spanish veterinarians before the Spanish-American War. During the Cuban revolution he was imprisoned by the Spanish but was not executed because of the intervention of the French Consul, who claimed that Dr. Lainé was a French subject. Released from prison, Dr. Lainé came to the United States where he worked strenuously to secure Cuban independence. At the outbreak of the Spanish-American War Dr. Lainé returned to Cuba as a war correspondent for the *New York Times*. After Cuba secured her independence he took an active part in efforts to elevate the veterinary profession and to improve the live stock industry of Cuba. Dr. Lainé was an expert horseman, a real sportsman and had hosts of friends.

N. S. M.

Our sympathy goes out to Dr. H. Meade Hamilton, of Muncie, Ind., in the death of his mother, January 29, 1928; to Dr. Crittenden Ross, of Middletown, N. Y., in the death of his father, March 14, 1928; and to Dr. A. E. Bott, of East St. Louis, Ill., in the death of his wife, April 7, 1928.

PERSONALS

MARRIAGES

Dr. L. H. Lewman (Colo. '21) to Miss Anna Louise Bubank, both of Big Springs, Nebr., February 18, 1928, at Sterling, Colo.

Dr. George J. Worner (Mich. '18) to Miss Myrtle M. Nelson, both of Bismarck, N. Dak., April 5, 1928.

BIRTHS

To Dr. and Mrs. Wm. R. Lukens, of Hillsboro, Ohio, a son, Teddy, February 29, 1928.

To Dr. and Mrs. A. C. Davidson, of Edwards, N. Y., a son, James Elton, March 4, 1928.

To Dr. and Mrs. W. N. Gaston, of Peterson, Iowa, a daughter, Marcia Anne, March 13, 1928.

To Dr. and Mrs. Harold J. Classick, of Hurley, S. Dak., a son, Leslie Glade, April 17, 1928.

PERSONALS

Dr. T. B. Huff (K. C. V. C. '08) has been elected Mayor of Sioux City, Iowa.

Dr. C. R. Dilts (O. S. U. '04) has removed from Fremont, Ohio to Napoleon, Ohio.

Dr. H. Gross (McK. '12) formerly of Ralston, Nebr. is now located at Laurel, Nebr.

Dr. Frank C. Wilson (Colo. '23) formerly of De Land, Fla., has removed to Leesburg, Fla.

Dr. W. Lenton (Ont. '05), who was formerly at Junor, Sask., is now at Medstead, Sask.

Dr. O. E. Gladfelter (U. P. '17) of York, Pa., has moved into larger quarters at 514 South George St.

Dr. W. H. Erwin (Ont. '97), of Howell, Mich., is building a new veterinary hospital to replace his old one.

Dr. L. H. Tripp (Corn. '18) is Albany County (N. Y.) Veterinarian, with headquarters at Albany, N. Y.

Dr. T. S. Burke (Ont. '18), of Windsor, Ont., has been appointed inspector for the Windsor Humane Society.

Dr. L. H. Conlon (Corn. '23), formerly of Vergennes, Vt., has requested a change of address to Towanda, Pa.

Dr. B. B. Loveland (Corn. '18) is Otsego County (N. Y.) Veterinarian, with headquarters at Oneonta, N. Y.

Dr. H. W. Eggleston (Corn. '14) is with the Borden Farm Products Company and is stationed at Alden, N. Y.

Dr. William L. Black (Colo. '24), who has been at Sonora, Texas, for several years, is now at State College, N. Mex.

Dr. W. A. Brunson (Corn. '18) has held the position of Health Officer at Daytona Beach, Fla., for the past five years.

Dr. J. T. McGrann (U. P. '21), of Trenton, N. J. is doubling the capacity of his veterinary hospital and boarding kennel.

Dr. Robert Simms (Corn. '11) is with the New Jersey Bureau of Animal Industry and is stationed at Morristown, N. J.

Dr. William M. Thomson (Corn. '16) is Saratoga County (N. Y.) Veterinarian, with official headquarters at Saratoga Springs, N. Y.

Dr. Andrew G. Vogt (Amer. '84), formerly located at Newark, N. J., is now a resident of Allenhurst, N. J. Address: 200 Elberon Ave.

Dr. W. R. Donahue (Corn. '23) is in the service of the New York State Department of Agriculture and is located at Sauquoit, N. Y.

Dr. P. J. O'Brien (R. C. V. S. London '07), of Los Angeles, Calif., is convalescing from an operation for appendicitis, performed in March.

Dr. G. S. Jermyn (Ont. '00), who has been located at North Battleford, Sask., has been transferred temporarily to work in Nova Scotia.

Dr. R. O. Biltz (U. P. '22), who is with the Pennsylvania Bureau of Animal Industry, has been transferred from Philadelphia to Harrisburg, Pa.

Dr. R. J. Fortney (U. P. '18), formerly associated with Dr. W. W. Hyde (Gr. Rap. '12), has opened a veterinary hospital in Wellington, Ohio.

Dr. S. H. Kesten (McK. '14), of the Health of Animals Branch, Canada Department of Agriculture, is engaged in area testing in Nova Scotia.

Dr. H. P. Roberts (O. S. U. '18), formerly of Edgeley, N. Dak,. recently accepted the position of Dairy and Food Inspector of Fargo, N. Dak.

Dr. H. H. Sheeler (Wash. '21), who has been located at Modesto, Calif., has requested a change of address to 320 California Ave., Watsonville, Calif.

Dr. A. J. DeFosset (O. S. U. '07), of Columbus, Ohio, recently addressed the Xenia (Ohio) Rotary Club on the subject of "Tuberculosis Eradication."

Dr. F. F. Bushnell (Corn. '02), in addition to his private practice at South Manchester, Conn., also finds time to serve as Meat and Milk Inspector of his town.

Dr. R. C. Calkins (Chi. '08), who has been practicing in Peoria, Ill., for a number of years, has made plans to open a modern small-animal hospital in Rock Island, Ill.

Dr. A. C. Davidson (Corn. '26), of Edwards, N. Y., entered the employ of Drs. E. J. and J. C. Robbins, May 1, at their new establishment at Blue Point, Long Island, N. Y.

Dr. Thomas B. Heim (O. S. U. '26), formerly of Alliance, Ohio, is now associated with Drs. Case and Planz, of Akron, Ohio. His mail address is R. D. No. 2, Akron. Ohio.

Dr. L. M. Lounsberry (Corn. '18), who has been connected with the Commercial Feed Department of the Pillsbury Flour Company for the past five years, is planning to re-enter private practice.

Dr. Frank Thorp, Jr. (Iowa '26), who has been at the Michigan State College for the past year, has accepted a position in the Division of Animal Pathology and Hygiene, University of Illinois, Urbana.

Dr. H. E. Kreidler (U. P. '21) has resigned his position with the Supplee-Wills-Jones Milk Company, of Philadelphia, and is now associated with Dr. J. T. McGrann (U. P. '21), of Trenton, N. J.

Dr. U. G. Houck (U. P. '95) holds the position of Associate Chief, Bureau of Animal Industry, U. S. Department of Agriculture, in addition to being Chief of the Hog Cholera Division of the Bureau.

Dr. F. A. Lambert, (O. S. U. '10), of Columbus, Ohio, recently underwent an operation for appendicitis. Latest available reports indicate that Dr. Lambert is making satisfactory progress toward recovery.

Dr. M. W. Drake (Amer. '90), one of the oldest practicing veterinarians in Philadelphia, recently closed his office at 1308 Morris Street, following the death of his sister. Dr. Drake is now living at 5623 Larchwood Ave., Philadelphia, Pa.

Dr. A. E. Wight (Harv. '97) has been promoted to the position of Chief of the Tuberculosis Eradication Division of the Bureau of Animal Industry, U. S. Department of Agriculture, succeeding the late Dr. John A. Kiernan (N. Y. C. V. S. '94).

Dr. E. F. Graves (K. S. A. C. '27), who has been spending some time at his home in Manhattan, Kans., as reported in the April number of the JOURNAL, has returned to his official position, as Territorial Veterinarian of Alaska, with headquarters at Juneau.

Dr. E. F. Sanford (Amer. '98), of Glen Cove, Long Island, N. Y., has been in St. Vincent's Hospital, New York City, for the past month, due to an infection in both arms. Dr. J. F. DeVine (Amer. '98), of Goshen, N. Y., has been looking after Dr. Sanford's practice during his illness.

Dr. F. D. Walmsley (Corn. '04), vice-president of Borden's Farm Products Company of Illinois, addressed the Veterinary College Forum, New York State Veterinary College at Cornell University, March 27, 1928, on the subject of "Veterinary Supervision of Commercial Milk Supplies."

Dr. G. W. Pope (Chi. '89) has been appointed Chief of the Field Inspection Division of the Bureau of Animal Industry, U. S. Department of Agriculture, succeeding Dr. A. W. Miller (K. C. V. C. '01), whose appointment as Chief of the Packers and Stockyards Administration was announced in the February number of the JOURNAL.

Captain Rafael Santa Maria (Havana '10), of the Cuban Army Veterinary Corps, has been appointed, by presidential decree, to organize a system of meat inspection for the Cuban Government. Dr. Santa Maria recently spent two years in the United States, studying the system used in this country, and is eminently qualified for the important work which he has undertaken.